NERVOUS AND MENTAL DISEASE MONOGRAPH SERIES NO. 9.

# Studies in Psychiatry

Vol. I

By

Members of the New York Psychiatrical Society

NEW YORK

THE JOURNAL OF NERVOUS AND MENTAL DISEASE
PUBLISHING COMPANY

1912

First reprinting 1970, Johnson Reprint Corporation

Printed in the United States of America

In the early winter of 1903, Dr. Allen McLane Hamilton invited a number of physicians to spend an evening with him for the purpose of discussing the advisability of founding a society for the promotion of the interests of psychiatry. This conference was held and, as a result of it, the constitution and by-laws of the Psychiatrical Society of New York were adopted on March 2, 1903. The Society has grown rapidly but has always retained its original semi-private character. The meetings have been held at the invitation of individual members, but any physician who has shown especial interest or achievements in psychiatry is eligible for membership. Since its foundation, four meetings a year have taken place, at which original contributions have been read and discussions carried on on various subjects connected with psychiatry. Many of the papers have been published, but until now no attempt has been made to collect them in permanent form. When the question of publication came up it was found that an issue of all the contributions would be too great an undertaking. So some papers were chosen for publication now, while others were left for subsequent volumes.

PEARCE BAILEY, *Chairman,*
ADOLPH MEYER,
GEORGE H. KIRBY,
*Publication Committee.*

# LIST OF MEMBERS OF THE NEW YORK PSYCHIATRICAL SOCIETY

Maurice C. Ashley ..... Middletown, N. Y.
Pearce Bailey ..... New York City.
Swepson J. Brooks ..... Harrison, N. Y.
C. Macfie Campbell ..... White Plains, N. Y.
L. Pierce Clark ..... New York City.
Henry A. Cotton ..... Trenton, N. J.
Charles L. Dana ..... New York City.
*Emmet C. Dent ..... New York City.
A. Ross Diefendorf ..... New Haven, Conn.
Albert Warren Ferris ..... Albany, N. Y.
Menas S. Gregory ..... New York City.
Allan McLane Hamilton ..... New York City.
Graeme M. Hammond ..... New York City.
August Hoch ..... New York City.
William Hirsch ..... New York City.
Smith Ely Jelliffe ..... New York City.
George H. Kirby ..... New York City.
Phillip C. Knapp ..... Boston, Mass.
Robert B. Lamb ..... Fishkill-on-Hudson, N. Y.
*Alexander E. McDonald ..... New York City.
William Mabon ..... New York City.
Carlos Macdonald ..... New York City.
Adolf Meyer ..... Baltimore, Md.
Flavius Packer ..... New York City.
Stewart Paton ..... Princeton, N. J.
Frederick Peterson ..... New York City.
William L. Russell ..... White Plains, N. Y.
Henry R. Stedman ..... Boston, Mass.

* Deceased.

# CONTENTS.

# THE INSANE IN JAPAN

By Frederick Peterson, M.D.,

PROFESSOR OF PSYCHIATRY, COLUMBIA UNIVERSITY, NEW YORK

During a vacation spent last summer (1909) in Japan, I visited a number of institutions for the insane, and through the many courtesies of Professor Kure and Professor Miura of Tokyo and Professor Imamura of Kyoto, I not only saw them under the best auspices but was furnished with much information in relation to psychiatry in Japan which I shall briefly put before you.

The medicine of ancient Japan, like its art, literature and religion, was derived from China by way of Corea. The earliest Chinese medical literature which deals in any manner with insanity dates from about 200 B.C. The earliest historical reference to insanity in Japan is contained in the law of about 702 A.D., which required the insane, epileptics, lepers, blind and crippled to be given over to certain official caretakers, who on taking such cases into their families were absolved from taxation and civic duties. Between these dates and for some time later Japanese physicians were guided in their study and practice wholly by Chinese medical books, in much the same manner as the Europeans for centuries acted only on the authority of Hippocrates, Galen and the Arabian writers. Insanity and epilepsy are well described in the first Japanese book of medicine, the "Ishinho," appearing in 982 A.D. For several centuries after this, medical treatment fell chiefly into the hands of the Buddhist priests who practised only with magic and prayer, until the period between the seventeenth and nineteenth centuries, when medicine reawakened and the Japanese physicians out-distanced in all respects their Chinese progenitors and contemporaries. The treatment of insanity during this period did not differ much from that of the more ancient day, and consisted chiefly of the sweat-cure, catharsis, emetics, thermocautery with moxa, hydrotherapy, acupuncture and at times blood-letting. The needle and moxa as

counter-irritants have for ages been favorites of both the Chinese and Japanese in all manner of diseases. Hydrotherapy, described in Chinese literature as long ago as 200 B.C., has always been a preferred method of treatment among the Japanese. Its use in insanity is described in the first Japanese book of medicine already referred to (982 A.D.).

In old times the insane were for the most part kept in families, the milder cases taking part in work on the land, or in the innumerable household crafts of that people. If subject to periods of excitement mechanical restraint was used, anklets, wristlets, chains, and solidly built chambers attached to the paper houses. Often in the country the patients were blistered on the soles of the feet to make them disinclined to run away.

A kind of family care grew up gradually, sometimes evolving into a colony system; and many private asylums were established long before any public asylums such as we have in the West were created.

Along in the early eighties the first public asylum in Japan was organized and established at the present capital, Tokyo. It was constructed somewhat on German lines, but with due regard to the necessities of earthquake architecture, for in a country where an earthquake is almost an everyday occurrence it is essential to build wisely. The Tokyo asylum consists of a series of one-storied pavilions scattered in a considerable park. The German traces in construction and arrangement are of course due to the fact that the foremost Japanese physicians of that day had taken their training in Germany, and the medical profession was wholly directed in all its undertakings by German influence. Nowadays with several universities of their own and a goodly number of medical faculties, quite equal to any in the world, in which all of the professors and the tongue spoken are Japanese, they need not go abroad for medical study.

In the older buildings of the Tokyo asylum the usual western corridor system prevailed, with numerous single rooms, but as time went on they began gradually to remove partitions and to convert the series of single rooms into good sized dormitories. This was the more readily possible because there seems on the whole to be less excitement among the Japanese insane than among the insane of other countries. The extraordinary quiet-

ness of asylum wards in Japan has been commented upon by other foreign visitors. It doubtless depends upon that immemorial training in the repression of emotional expression which is so noticeable a feature in Japanese psychology.

In some of the newer pavilions they have European bedsteads, tables and chairs, but for the most part the furnishing is Japanese, thick mattings upon the floors, thick quilts laid upon these mattings for beds and tiny dwarf tables when such are needed, with no chairs or other furniture.

The asylum is lighted by electricity, electric lighting being a specialty of the Japanese everywhere at present, owing to unlimited waterpower in innumerable mountain torrents which have been harnessed to do this work. There is provision for the daily hot bath for every inmate according to Japanese custom, for every man, woman and child in Japan takes at least one hot bath a day, and sometimes two or three; and our western systems of plumbing for bath and toilet purposes have been adopted and installed. There is provision also for the prolonged bath which has so much vogue with us at present in the treatment of the insane.

Much is made of occupation. The laundry and garden work are done by inmates. There were rooms in which patients were weaving, plaiting straw, making paper envelopes, and carrying on other crafts.

All the buildings were airy, neat and clean, and to me a striking feature of the care of the insane was the morale of the nursing staff. I believe such gentleness, kindness, patience, and assiduous attention to the sick could be found nowhere else, for nowhere else exists a whole race of people who never scold, quarrel or manifest impatience, but always turn a smiling face and extend a helpful hand to one another. This other fact in Japanese psychology I observed among all classes throughout my visit. It was particularly noticeable where I least expected it, among the lower classes.

The insanity clinics of the university are held here and the laboratories of the asylum are well-equippd with pathological and psychological apparatus. Elaborate histories of the patients are taken and besides the director, Professor Kure, there are ten physicians working in the hospital whose capacity is about 500

beds—a capacity of one to fifty. We are lucky in New York State to have one physician to two hundred patients.

There can be no overcrowding in the Tokyo asylum, for according to law a new patient is only admitted when there is a vacancy. An indigent patient brought before the authorities is sent at public expense to one of the seven private asylums in Tokyo if the government asylum has no bed. These seven private asylums have a capacity of about one thousand beds between them.

The method of commitment is simple. When a case of insanity develops, a member of the family reports it to the police. A doctor then goes to the house with the police officer to examine the patient and reports his findings to the head of the police who issues an order of commitment. In the country a governor's certificate takes the place of police commitment. There is never any effort on the part of patients to escape.

The Tokyo institution has one of eleven psychiatric clinics in Japan. I believe we have not so many in the United States. There are psychiatric clinics at the three universities Tokyo, Nagasaki and Fukuoka, and at the medical schools of Okayama, Kawazawa, Kyoto (two medical colleges), Nagoya, Shibu, Sendai and Osaka.

As regards the character of the cases observed in Japan, there are several interesting points. In the first place the classification approximates very closely to ours and we observe large numbers of cases of dementia præcox, manic-depressive insanity, general paresis, and the like. True paranoia as we know it is a great rarity. On the other hand general paresis and dementia præcox are more common than with us. At the Tokyo asylum the proportion of cases of general paresis from the years 1887 to 1901 was 15.86 per cent.

In a country which has no opium or alcohol vice, inebriety cases are rare. Only 6.65 per cent of the admissions of men presented mental disorder due to alcohol. A case of alcoholic insanity in women is almost unknown. Very few doctors in Japan have even seen a case of delirium tremens. Korsakoff's psychosis has not been observed. The alcohol cases are due to sake, a mild kind of sherry-flavored wine, derived from rice. Only the lower classes drink sake to excess, and very few of

these. Temperance societies are growing rapidly in Japan. In Formosa, a province of Japan, considerable opium is used, but scarcely any in Japan itself. There are no cases of Indian hemp or cocaine inebriety. I noted a considerable number of cases of insanity wholly new to me, viz: psychoses associated with Kakke or beri-beri. The multiple neuritis and mental symptoms made a picture something like that of Korsakow's psychosis. In our western books on nervous diseases, mental symptoms are not described at all as associated with beri-beri, but in Japan we have a beri-beri psychosis which reminds one partly of pellagrous insanity and partly of the Korsakow syndrome.

I come now to the most interesting part of my journey of observation in Japan.

About seven miles from Kyoto, one of the ancient capitals of Japan, lies the village of Iwakura, to which one day Professor Imamura, professor of Psychiatry in the University of Kyoto, conducted me. It is alone well worth going to Japan to see. I believe I am the second westerner who has been there, the first having been Dr. Stieda of Russia who mentions it, and calls it a Japanese Gheel in an article on "Psychiatry in Japan" in the Centralb. f. Nervenh. und Psychiatrie for 1906. Professor Imamura has himself described it in the Transactions of the International Congress of Psychiatry, Neurology and Psychology, Amsterdam, 1907. But it deserves to be better known, for as an ideal place for the care of the insane it is unique, there being only one other institution for the insane that I know of that in anyway embodies what should be our own ideal as far as concerns surroundings and construction, and that is the Maison de Falret at Vanves in the outskirts of Paris.

The third daughter of the Emperor Gosanjo in the eleventh century developed melancholia in her eighteenth year. Word was brought to the imperial household that at Iwakura was a holy fountain the water of which was healing to mental diseases and to disorders of the eyes. The Emperor's daughter was taken there nearly 900 years ago and recovered and so brought fame to the temple and the well of Iwakura, as a result of which the insane were brought there in great numbers. At first three small inns were constructed to receive them, then later tea houses and villas and cottages sprang up in which to care for the ever increasing influx of patients.

In the year 1889 the village had 239 houses, with 1,579 inhabitants, and up to that year one to two patients were received into each family to share in the occupations of the household which were chiefly out of door employments in fields, gardens and forests.

The village lies at the foot of great hills, in a beautiful wide valley. The hills are covered with evergreen cryptomeria pines and spruces, while the valley is cultivated every foot of it with rice and vegetables. Each little house has its own idyllic charm. This charm lies in its simplicity of architecture, harmony with the landscape, and in its well-studied gardens both inside the court-yard and outside around the house. Paper windows and removable paper walls insure light and air and a practical out-of-door life night and day. From the spotless mats upon the floor across the spotless verandahs one looks out upon the gardens green with pines and cedars all the year round, with flowering shrubs for every month between the winters, looks out into the restful gloom of the giant cryptomeria woods on the one side, or across the valley of rice-fields to the evergreen hills upon the other. There are beautiful paths and roads among these cedar forests, and several imposing temples among them.

In 1889, the Japanese government, evidently under the impression gained from a study of the asylum systems of Europe and America came to the conclusion that their colony system that had grown up so naturally was too far from our western ideals, as exemplified in our colossal caravanseries for the insane, and so forbade the insane being any longer taken to the village of Iwakura. They abolished the method as probably barbarous, just as at one time they abolished cremation, having been persuaded by Europeans that it was heathen practice, but returned to it again when they learned that cremation was the goal to which western civilizations are tending in the method of disposal of the dead.

The result of this opposition of the government has been to reduce at least temporarily the number of insane in the colony. It is altogether likely that as soon as the authorities learn that out of themselves they have developed through nearly a thousand years the best of all methods of caring for the insane, toward which the West itself is struggling with much difficulty, they will remove the proscription and restore Iwakura to its ancient

rights and privileges under state organization and inspection. There is one retreat for about 90 patients at Iwakura built on European models under the care of physicians, to which excitable cases may be brought from the family homes in the neighborhood.

I have already referred casually to the Maison de Falret, a French asylum in the suburbs of Paris, and in connection with the results of my visit to Japanese institutions I cannot forbear to mention it again and to say a few words about a place that is probably unknown to most of my hearers, because after all the chief value of any observations by a traveller must be the new knowledge, the example, the lesson or the moral that he brings home to his own people. Doctors Voisin and Falret two specialists in psychiatry nearly one hundred years ago, purchased an estate of over sixty acres in the environs of Paris, made of it a park, and planted it well with trees and shrubs. A pretty stream courses through it. They built therein small houses or bungalows, each surrounded with high green hedges and pretty gardens with its own gateway. Now after a hundred years it realizes their dream of what should be done for the insane. It is a large park with magnificent trees and shrubbery, divided into two halves by a farmstead group, thus making practically two parks, one for each sex, and there are twenty-seven such bungalows for the isolation of one or more patients. A patient here is not only isolated from his friends, which is usually a distinct advantage, but is isolated from the insane, which is an even greater gain. I cannot take time to describe it here, but it shares with Iwakura in Japan the distinction of being an ideal retreat from the standpoint of environment and construction for mental cases.

We find then in Japan and in France a certain standard already attained and realized, which we might take for our own. These two places leave nothing to be desired in the way of surroundings and method of construction. We should perhaps be able to add something of our modern machinery to these plans, in the way of central heating, organization of food supply and service, telephonic intercommunication, and latter day hydrotherapy, but these are not the essentials. The essentials for the care of the curable insane are already here, and these are adequate nursing, segregation, the return to nature, the simple life,

beautiful surroundings, association with normal and not insane persons, and plenty of space and opportunity for walks, for working in gardens and fields and at various arts and crafts.

Have we already drifted too far from the realization of Vanves and Iwakura, with our vast aggregations of 3,000 to 5,000 patients in one institution, with the sinking of the individual in the mass, with our appalling overcrowding, with our inferior nursing staff and insufficient medical staff, with our at best rudimentary methods of occupation, and with our immense, expensive and complicated machinery of mere support and custody?

# A STUDY IN RACE PSYCHOPATHOLOGY

By George H. Kirby, M.D.

DIRECTOR OF CLINICAL PSYCHIATRY, MANHATTAN STATE HOSPITAL, WARD'S ISLAND, N. Y.

Studies in psychopathology which seek to analyze and compare the abnormal mental states found in the different branches of the human race touch a number of highly interesting topics and have an important bearing, not only on the special problems of psychiatry and mental hygiene, but they also promise to furnish valuable data for educators and social workers.

Just as we see racial traits and peculiarities of a people finding expression in their normal mental activities, in their religion, morals, politics and artistic productions, so we may expect to discover that racial characteristics are imparted to the abnormal mental life, modifying or coloring the clinical forms of those psychoses common to the different ethnological groups of mankind.[1] But our inquiry leads us further than the study of how the form and symptoms of a psychosis may vary in different races; the deeper and more important question of etiology becomes the principal consideration when it is shown that one race is more liable than another to suffer from a certain kind of mental disease. The cause for this susceptibility of one race and relative immunity of another can only be explained fully when a whole series of complex factors has been analyzed.

In the large group of mental disorders dependent on exogenous causes, such as syphilis, alcohol, infectious diseases or other physical disturbances, we see clearly the important rôle played in the genesis of these psychoses by the sexual life, social customs, occupations and habits of the race.

In the other large group of mental disorders, the so-called functional psychoses, endogenous etiological factors seem to play the

[1] See an interesting report by Professor Kraepelin on the Mental Disorders of the Natives of the Island of Java. Centralblatt für Nervenheilkunde und Psychiatrie, Vol. 15, 1904, p. 433.

most important rôle and among these are to be mentioned in the first place certain general tendencies of the personality, analysis of which allows us to differentiate several types of mental makeup. My interest in this investigation was first awakened by Meyer's description of the various types of personality and constitution, and their meaning for psychiatry.[2] Interest was further stimulated by Hoch's recent communication on types of mental makeup and their relation to the functional psychoses. In the light of these studies the subject of race psychopathology becomes especially important. If groups of individuals, because of a peculiar kind of mental makeup, are prone to develop a certain form of psychosis, then in the larger racial divisions which present such distinctive types of character and personality, we may also expect to find that certain forms of mental disturbance predominate in the one or the other race. To understand this racial tendency through analysis of the factors operative in the inner mental life of a people would mean to make an important addition to our knowledge of the development of the functional psychoses.

Most of the studies hitherto made in comparative psychiatry are of little value. This is the natural result of the lack of uniformity in the clinical conceptions of different observers, together with the confusion in nomenclature. It is, therefore, to-day utterly useless to attempt to use the hospital reports of different countries in order to estimate the frequency with which any particular mental disorder occurs in the various races, or to learn what deviations, if any, exist in the clinical forms of the psychoses occurring among the different peoples of the world. For investigations in this field to be of any value, it would seem essential, as pointed out by Kraepelin, that the studies be carried out by the one observer; otherwise, no comparable data are to be expected. This requirement is readily met at the Manhattan State Hospital where the clinical material offers a rare opportunity for research in comparative psychiatry. In New York city the conditions of a nice experiment are practically fulfilled in that a number of races of pure blood are found living in large colonies in a uniform general environment. The outcome of such a situation must be full of interest to the psychopathologist.

[2] An Attempt at Analysis of the Neurotic Constitution. Adolf Meyer, American Journal of Psychology, Vol. XIV, p. 90, July–September, 1903.

In this preliminary report I wish to present merely the result of a review of the clinical material of the past year[3] made to ascertain the relative frequency of the different psychoses in the various races entering the hospital. Among the admissions during the year were found representatives of twenty-seven different racial groups. In seven of these groups the number of cases seems sufficiently large to allow certain comparisons. Our attempt has been made to compare in the first place *racial stocks,* without regard to nationality or geographical distribution. Under the Irish, for instance, we include native-born Irish and the first generation of children born in America of native Irish parents. In a similar way the German, Italian and English groups are formed. We have restricted the American group so as to comprise only those indivdiuals whose parents were born in the United States, but this does not include the Negroes and Jews, who are kept apart and form each a group without regard to country of birth or length of time in America.

| Psychosis. | Irish, Per Cent. | Jewish, Per Cent. | German, Per Cent. | U. S., Per Cent. | Italian, Per Cent. | Negro, Per Cent. | English, Per Cent. | All Other Races, Per Cent. | Total No. in Each Psychosis. |
|---|---|---|---|---|---|---|---|---|---|
| Psychoses with organic nervous disease | 2.69 | 0.98 | 1.54 | 2.38 | — | 1.96 | 2.85 | 1.53 | 25 |
| Senile psychoses | 9.80 | 2.87 | 6.70 | 7.14 | 3.70 | 9.80 | 5.71 | 5.64 | 92 |
| General paralyses | 7.59 | 14.05 | 20.10 | 17.46 | 9.87 | 29.41 | 14.28 | 10.25 | 184 |
| Alcoholic psychoses | 27.69 | 0.32 | 11.85 | 11.90 | 8.64 | 7.82 | 11.42 | 7.69 | 182 |
| Infective-exhaustive | 5.39 | 4.47 | 8.64 | 4.76 | 8.64 | 5.88 | — | 6.66 | 74 |
| Involution melancholia | 1.71 | 3.19 | 2.06 | 1.58 | 1.23 | — | 2.85 | 1.53 | 28 |
| Depressions undifferentiated | 1.96 | 5.43 | 5.15 | 1.58 | 8.64 | 3.92 | — | 9.22 | 64 |
| Dementia præcox | 13.48 | 27.47 | 14.95 | 16.66 | 23.44 | 13.72 | 28.57 | 29.23 | 284 |
| Paranoic conditions | 5.14 | 1.59 | 8.25 | 7.92 | 7.40 | 5.88 | 8.57 | 5.12 | 74 |
| Manic-depressive | 16.66 | 28.43 | 12.89 | 18.25 | 13.58 | 9.80 | 17.14 | 13.33 | 253 |
| Epileptic psychoses | 2.20 | 1.59 | 4.64 | 3.17 | 4.93 | 3.92 | 2.85 | 1.02 | 36 |
| Constitutional inferiority and psycho-neuroses | 2.94 | 6.07 | 4.64 | 3.96 | 2.46 | 1.96 | 5.71 | 2.56 | 53 |
| Idiocy and imbecility | 0.24 | 0.98 | 0.51 | 0.79 | 1.23 | — | — | — | 7 |
| Unclassified | 2.45 | 2.55 | 2.06 | 2.38 | 6.17 | 5.88 | — | 6.15 | 45 |
| Total number persons each race | 408 | 313 | 194 | 126 | 81 | 51 | 35 | 195 | 1403 |

The accompanying table shows the results of the analysis. The total number of patients included in the study was 1,403. The bottom row of figures shows the number of each racial type admitted to the hospital, the Irish with 408 persons forming the

[3] Cases admitted from October 1, 1907, to September 30, 1908.

largest group, the English with 35 persons forming the smallest. The vertical columns show the percentage distribution of the various psychoses within each racial group.

Irish stock furnished nearly 30 per cent. of all the admissions. The figures for this race demonstrate in a most convincing manner the important rôle played by alcohol in the mental disturbances of the Irish people. Twenty-seven per cent. of all the Irish admitted were suffering from alcoholic insanity, the proportion being more than *double* that found in any other race. Within the alcoholic group itself the Irish contributed *62 per cent. of all the cases* (113 out of 182 cases of alcoholic insanity). Accompanying this extraordinarily large percentage of alcoholic disorders we find further that the Irish stand highest in senile dementia and psychoses accompanying organic nervous diseases. In general paralysis, on the other hand, the percentage is lower than in any other race. This latter finding was rather a surprise. It is an interesting fact taken in connection with the view often expressed, that alcohol plays an important rôle in conjunction with syphilis in the causation of general paralysis. Our figures for the Irish race tend to show a closer relationship between alcoholism, senile dementia and various organic brain diseases than between alcoholism and the meta-syphilitic disorders, such as general paralysis.

The figures for the Jewish race bring out several interesting facts. One notices first of all that the Hebrews are practically free from alcoholic psychoses. The figure .32 per cent. represents a single case which occurred in a series of 182 cases of alcoholic insanity. I must also add that this particular patient, a man, is still under observation, having been over a year in the hospital and certain features in the development of the psychosis as well as the course of the disorder suggest the possibility that the case may after all belong with the paranoid dementias. We notice the further interesting fact that the absence of alcoholic insanity in the Hebrew is accompanied by the lowest figure for senile dementia and psychoses with organic nervous diseases. The next most noteworthy fact gathered from the second column is that the Hebrew race shows by far the greatest percentage of manic-depressive cases (28.43 per cent.) and the Jew also stands highest in the psycho-neuroses and constitutional inferiorities and in involution melancholia. In dementia præcox, with the exception of

the English people (28.57 per cent.), the Hebrews are again foremost (27.47 per cent.). In the undifferentiated depressions they are next to the highest. We thus see that in the large group of the so-called functional psychoses, by which we mean those disorders in which certain endogenous or psycho-genetic factors seem most important as upsetting causes, the Jewish people outnumber enormously any other race.

Among the Germans general paralysis ranks high (20.10 per cent.), a higher percentage being reached only in the Negro (29.41 per cent.). Mental disturbances of alcoholic origin are also rather frequent in the Germans, and one observes a striking uniformity in the figure for the Anglo-Germanic group—the German, American and English groups each showing 11 per cent. of alcoholic psychoses. We notice further that the Germans are also relatively high in senile psychoses, infective-exhaustive states, paranoic conditions, and the psycho-neuroses and constitutional inferiorities. In the manic-depressive group the Germans rank low (12.89 per cent.), the only lower figure being found among the Negroes (9.80 per cent.).

The figures for descendants of native born Americans are given in the fourth column. The percentages in this group seem to occupy somewhat of an intermediate position between those of the other races, that is to say there are no extraordinarily high or any strikingly low figures in the American group. Manic-depressive insanity is, however, rather high, a higher percentage being found only in the Hebrew; in alcoholic psychoses and general paralysis a relatively high figure is reached by the native Americans.

In the remaining three races—Italian, Negro and English—the number of cases is smaller than in the preceding groups and the percentages are therefore probably less representative for these races.

In the Italian group we find that general paralysis and alcoholic psychosis are both strikingly low. The undifferentiated depressions and dementia præcox are high. *Epileptic disorders are more frequent than in any other race (4.93 per cent.).* We see further that the unclassified group shows a high figure which may mean that the Italian people offer a larger number of atypical or obscure disorders than other races. It may be, however, that inaccessi-

bility because of the language difficulty is partly responsible for this high figure.

In the black race we meet with a remarkably high percentage of general paralysis (29.41 per cent.), higher by far than that found in any other race. This figure may surprise one in view of claims not long since made that the Negroes were almost entirely free from meta-syphilitic disorders, not only general paralysis but also tabes. The proportion of women among the Negro general paralytics seems to be unusually high. The average of all races exclusive of the Negro was not quite 4 men to 1 woman. In the Negroes we find the proportion to be 3 men to 2 women. The alcoholic disorders are lower in the Negro than in any other race except the Hebrew. This low proportion of alcoholic insanity was hardly expected, as the Negro has been described as being especially sensitive to toxic influences. Manic-depressive insanity seems to be infrequent in the Negro, the percentage (9.80) being in fact lower than that found in any other race.

Among the English patients, of whom there were only a small number admitted, 35 altogether, we find that dementia præcox is proportionately more frequent than in any other race (28.57 per cent.), the figure being, however, only slightly higher than that found in the Hebrew (27.47 per cent.).

*The more important results of the study may be summarized as follows:*

The Irish are clearly more prone to develop alcoholic disorders than any other one of the races considered. They are also more liable to senile deterioration and other psychoses with organic brain disease.

The Jewish race seems practically free from alcoholic insanity. The Hebrew, however, ranks higher by far than any other race in the functional group of psychoses made up of manic-depressive insanity, dementia præcox, constitutional disorders and depressions of various form.

In the Negro general paralysis occurs proportionately with more frequency than in any other race. Alcoholic disorders remain at a low figure. In the functional psychoses, particularly manic-depressive, the Negro ranks low.

The Germans are relatively high in general paralysis.

The Italians are low in both general paralysis and alcoholic

insanity. They are highest in epileptic disorders and furnish the largest percentage of unclassified cases.

The English are highest in dementia præcox but the small number of English people included in the study renders this figure rather unreliable.

The American group shows no striking figures when compared with other races, but manic-depressive insanity, general paralysis and alcoholic disorders reach a relatively high percentage in people whose parents were born in the United States.

# THE CURABILITY OF EARLY PARESIS

By Charles L. Dana

PROFESSOR OF NERVOUS DISEASES, CORNELL UNIVERSITY MEDICAL COLLEGE, N. Y.

## Introductory

In Professor Tanzi's recent work on Mental Diseases, discussing progressive paralysis, he states that the disease may be unrecognized for years and that the actual duration of paresis may be ten or fifteen years—in fact may last as long as do cases of tabes.

With regard to the prognosis, he says:

"Although it is dogmatically stated that progressive paralysis is essentially an incurable and fatal disease, we can not entirely exclude that if it is promptly diagnosed and energetically treated, the morbid process may be arrested. Moreover, the essentially initial lesions of progressive paralysis (chromatolysis of nerve cells, tumefaction of cell body) are not of a destructive character, nor are they altogether irreparable" (Tanzi, Mental Diseases, 1909, p. 419).

Dr. Gilbert Ballet recently reported a case (Sociètè de Psychiatrie, L'Encephale, February 10, 1911) of a patient who in 1902 or 1903 presented very marked delusions of grandeur with symptoms of tabes. The patient recovered from his mental disturbance and is now well. Ballet considered him to belong to a group of cases to which he has called attention, of general paralysis with possible arrested evolution, "paralysis générale à evolution discontinué."

These are practically the views for which I have contended in the papers, the first being published seven years ago: viz. that so-called luetic neurasthenia, luetic melancholia and luetic pseudoparesis are often if not always, preliminary and preparatory stages of paresis. If energetically treated, the patient is cured or relieved, if not treated, the malady usually goes on into the degenerative and incurable stage. It occasionally happens that a

periodic functional psychosis (mania or melancholia) may be grafted upon a nervous lues.

The view that cerebral lues is one disease and paresis quite another is too narrow a one to hold in the light of recent observations.

In an inflammatory process we do not call the initial congestion and exudation one disease, the terminal cell degeneration and necrosis another, though one is curable, the other is not. So it seems to me that a cerebral lues with neurasthenic and mild paretic symptoms represent the beginning yet curable phase of a process that naturally and untreated ends in a hopeless degeneration.

Some interesting facts regarding the essential unity of the processes have been furnished by the newer methods of studying the blood and cerebrospinal fluids.

So far, the laboratory observations have not furnished any sure criterion by which we can say that a case is nervous lues, or is paresis. In each condition, there is an increase of cells in the cerebrospinal fluid, though less in paresis than in exudative syphilis of the brain; in each the blood and cerebrospinal Wassermann tests and the globulin tests are positive,—more intensely so in paresis, as a rule.

Dr. Kaplan has shown that under mercurial treatment the reactions of the cerebrospinal fluid in tabes, paresis and cerebrospinal lues respond in the same way. In all three of these conditions there are found before treatment:

Lymphocytosis +
Globulin test +
Blood-Wassermann +
Cerebrospinal Wassermann +
Fehling's reaction o

Under treatment, there comes usually earliest a reduction in the number of cells; then the globulin reaction becomes negative, then the blood-Wassermann and finally the cerebrospinal Wassermann tests also become negative. As the lymphocytosis diminishes the Fehling reaction returns. These reactions suggest that we are attacking the same conditions; and not as I believe three different diseases.

So far as investigations yet have gone, then, it seems rather plain that paresis is not a disease sharply split off from cerebral lues but rather a later phase of it. The exact line can not yet be drawn, but I contend that there is often a long period of time during which symptoms occur that are "pre-paretic," and that this condition should be recognized since it is curable, in the sense that it is kept as permanently under control as lues itself can be kept.

The phases in which pre-paresis shows itself most commonly are: (1) Luetic neurasthenia, (2) luetic neurasthenia with paretic symptoms (pseudo-paresis) (meningeal and cortical lues), (3) luetic melancholia and mania.

The forms of cerebral and spinal lues with local exudates, gummata, etc., may or may not be associated with or become precursors of paresis.

My experience teaches me that spinal exudative syphilis (in the common form of Erb's paralysis) is rarely followed by cerebral but often by spinal degenerations; cerebral syphilis with exudates are not ominous if they involve the brain stem; but the diffuse meningeal lues involving probably the cerebral cortex causes symptoms resembling paresis and leads directly to it unless treated. In this condition the symptoms sometimes can not be distinguished from the symptoms of paresis; and if neglected the evidences of degeneration in time almost invariably appear.

FIRST PAPER, READ NOVEMBER, 1904

I wish to present what to my mind is convincing evidence that paresis, in its very earliest stages, in that stage which may be called one of "preparesis," is a disease that sometimes can be arrested. This arrest may be permanent, and may be attended with so little mental defect that one may call the patient practically cured.

*Paresis, Tabes and Syphilis.*—It is now quite generally admitted that paresis is almost always a parasyphilitic disease; that is to say, one which is due to the late and degenerative influence of a luetic poison. Paresis is also looked on as a disease which has the same relation to the brain structures that tabes dorsalis has to those of the spinal cord.

This relationship, indeed, is so often and so plainly observed that it can be considered a proved clinical and pathologic fact. We find for example, in from 5 to 10 per cent. of the cases of degenerative syphilis of the nervous centers that the patient suffers both from paresis and from tabes, and has what is termed "tabo-paresis," the paresis gradually associating itself with tabes, or *vice versa.*

*The Arrest of Tabes.*—Now, nothing is more clearly established than the fact that tabes dorsalis is often arrested in its early stages, so that a patient may live for ten, twenty or thirty years, and exhibit no practical progress or change in his symptoms. I have a number of patients under observation who illustrate this undoubted condition; and my experience, I am assured, is the common one.

If, then, tabes may be arrested in its early or pretabetic stage, there seems no reason to suppose that we can not also arrest and cure paresis in its earlier stage, and this is what I believe can be done.

The cases which I report, taken in connection with other clinical experiences which it is impossible to present without making my paper too long, have been sufficient to make me feel quite sure of my position in the matter. I am not asserting that paresis, when it is once well established, can be cured. In fact, I do not think it can be; and I know of no more hopeless malady when it has once got a full start.

Paresis not infrequently shows remissions, and these remissions may be prolonged to one or two, or even to five or six years. In these remissions, however, the mind, by no means, is restored to its original tone or vigor. The patient has only a "let-up," and is never the sound, vigorous-minded man he was previously. My patients have been more than cases of remission; their condition of mind and body has been restored, practically, to a normal level. I am not asserting the existence of remissions in paresis; that is one of the admitted features in the natural history of the disease. What I do assert is that in some cases in which the patient has shown unmistakable evidences of a degenerative and paretic process starting in the brain, this process has been arrested, evidences of it have even sometimes entirely disappeared, and the patient has gone on with his usual work.

I have not yet had patients under observation for a sufficiently long period of years to enable me to say that the paresis will never return. I can only argue from analogy in the cases of tabes dorsalis, and since we know that here the arrest of progress is sometimes permanent, we may legitimately infer that when it has occurred in paresis in the same way it may also be permanent. I would say, further, that there is nothing *a priori* impossible in the idea that paresis may be arrested and cured when it first starts in. We are able to arrest degenerative processes in other parts of the nervous system; we arrest degenerative processes, or we see them arrested, in the kidneys, in the liver and in other organs. Given a vigorous constitution poisoned with disease, it may well be that when it is put under the best possible conditions for fighting this poison, when its known antidote has been administered with heroic thoroughness, we might expect that the tendency of the tissue to die may cease.

*Pseudoparesis.*—Also, in speaking of paresis, I wish to be understood that I am speaking only of general paresis or paralytic dementia, not of the so-called "pseudoparesis" of alcoholics or the "pseudoparesis" of syphilis. I have used these terms personally, and I know they are widely adopted as convenient expressions. I do not think, however, that this term, "pseudoparesis" is a very fortunate one, inasmuch as the disorders above mentioned are essentially simple forms of organic dementia, with an entirely different pathologic basis and clinical course from the real paresis. The so-called pseudoparesis of alcoholism, for example, is only an organic dementia, due to the connective tissue proliferation, the vascular changes, and the cellular atrophy brought on by the continued use of alcohol. The pseudoparesis of syphilis is simply a dementia brought on by the exudates of syphilis, leading to more or less severe secondary changes in the meninges and in the vascular supply. It is really an organic dementia, due to an exudative inflammation of the meninges and blood vessels. In true paresis the organic changes are comparatively slight at the beginning, and are probably mostly of a parenchymatous nature. There is no exudate and no early gross organic change. It is true, however, that in the early stages of paresis there may be some slight amount of luetic exudate, and that perhaps the degenerative changes in the cell are started by this process. We know

also that there are cases of paresis in which there are both true primary degenerative changes and real syphilitic exudates present at the same time, so that the patient may be said to be suffering both from brain syphilis and from a paresis. It is strictly analogous to the conditions which occur in a spinal cord, where at times there may be a true tabetic degeneration and at the same time a decided syphilitic exudate, so that we have both locomotor ataxia and spinal syphilis together.

It is at this point that the weakness of my case, as I freely admit, may lie. It may be and probably will be contended that the cases which I assert are in the stage of "preparesis" are really cases only of slight exudative brain syphilis, and that my patients have simply been cured of a slight degree of a perhaps rather diffuse vascular and meningitic exudate. To this I can only reply that I have many times seen precisely this class of cases passing directly into a condition of true paresis, and that in all of my cases where there were admittedly some symptoms of syphilitic exudate, or some kind of gross organic lesion, there were with it also decided psychical and somatic symptoms, such as occur only, or mainly, in connection with the degenerative process of paresis. An Argyll-Robertson pupil, for example, is the sign of the onset of a degeneration, not of exudation. At the very most, while admitting that my opponents may be academically right, I would claim that they are practically wrong, for the reason that, basing my argument on experience with other cases, I feel sure that all or nearly all of these patients would have gone into a condition of true paresis if they had been let alone. Thus, when a patient who has given a distinct history of syphilis develops a form of agitated melancholia, and at the same time shows signs of cerebral degeneration, like the Argyll-Robertson pupil and disturbances in the knee reflexes, I should certainly be apprehensive that, under ordinary conditions, he would eventually develop a paresis, for I have seen a number of patients who entered paresis through this peculiar gate of melancholia, with somatic signs as indicated.

Again, if a patient with a history of syphilis, after a certain period begins to develop convulsions and shows Argyll-Robertson pupils, exaggerated reflexes, then begins to develop symptoms of loss of memory, change of character and disturbances of the

instinctive feelings; I should feel very certain that if left alone he would pass into the condition of paresis, for it is through the gate of convulsive disturbances, epileptiform seizures and peculiar somatic signs that paresis sometimes develops.

Still further, when a patient, who gives perhaps a doubtful history of syphilis but whose life is such that he might easily have been subjected to it, and who has a headache, an eye-palsy, and previous to that has for some time shown great extravagance in action and ideas, with a decided change in character and weakness of memory, I would here also feel very sure that a paresis was developing.

Having observed the total disappearance of all these symptoms, under treatment, and the restoration of the patients practically to their former health, it has seemed to me that I may be right in claiming that it is possible to arrest for an indefinite time a disease which is certain to become a general paresis.

With these preliminary remarks, I submit the following records. They are not published in every detail because it seemed to me not necessary. I have given the salient facts with regard to the symptoms and course; besides this, the patients were all private patients of my own or of physicians who referred the cases to me, and some of the patients were men of prominence, whose intimate lives and identity I should dislike to expose. The histories should perhaps carry greater weight for the reason that they are not records of hospital or dispensary cases, in which data are often uncertain and the mental development of the patients of mediocre type:

## Case Reports

Case 1.—M. S., aged 40, married; occupation, broker.

*History.*—Family history shows a very bad indirect heredity. The patient had an infection before he was 20, which was treated. He had had seven healthy children. He had led a life of much social activity and business excitement. Three months before I saw him he began to get ideas of poverty and self-reproach, and when I saw him he had a distinct melancholia.

*Examination.*—The pupils were unequal, the left larger than the right; both distinctly Argyll-Robertson in type, though the right reacted very feebly to light. He claimed, however, that the left pupil had been larger than the right for fourteen years, and that this condition was due to a sunstroke (?). The left knee jerk was lively, the right weak. He had

some tremor of the hands, but none of the face. There was no speech disturbance. He had had no seizures. Mentally he showed no dementia, but only a very profound and anxious depression, with some delusions of poverty and self-accusation. He suffered from insomnia, but had no headaches, and had no cranial nerve palsies except those of the eyes, and no disorders of the spinal centers or ataxia.

*Treatment and Result.*—He was seen by two physicians, one a very competent neurologist, who thought that he was developing paresis. He was sent to a sanitarium, where he was put under active treatment. In six months he came back practically well. The pupil of the right eye had become normal; the left was the same as before; knee jerks same as before. He has continuted well and has been in active business now for over three years.

Case 2.—J. D. H., aged 37, married; merchant by occupation.

*Family History.*—Direct heredity good; one sister had epilepsy; no other neuroses in the family.

*Personal History.*—He had a luetic infection twelve years ago. He was a man of temperate habits, but worked extremely hard. About two years before I saw him he began to run down and complained of sensations of pressure on the side of his head, which symptoms continued very persistently.

*Examination.*—The pains and paresthesias extended down the back of the neck and into the shoulders. In other words, he had the annoying head, neck and shoulder paresthesias seen in beginning paresis. As his condition grew worse he began to be drowsy, and apparently required more sleep than normal. Three weeks before I saw him he had had two convulsions of an epileptiform character. At that time he was emotional and depressed, crying easily, and having apprehensions about his mind giving way. When seen by me he had been taking for some days 15 grains of bromid in the morning and 6 grains of trional at night. At this time he was depressed, his memory was poor, his spech was syllabic, but he had no tremor, and the pupils were normal, as were also the knee jerks. He was then having attacks of excitement at intervals with, at times, some brief delusions, but no hallucinations. He complained of his head and was very depressed and apprehensive. He appeared to me to have the physiognomy of a patient likely to develop paresis, and the history of convulsions, his speech disturbance, his feeble memory and his melancholia, all pointed in that direction. At the time I made a diagnosis of paresis, with a question mark.

*Treatment and Result.*—The patient was sent to the Watkins sanitarium, where he remained for several months under the care of Dr. King. He had one more convulsion. He returned much improved and gradually recovered. He has continued well to the present time, which is four years from the period when I first say him.

Case 3.—B. R., aged 34, married; occupation, business. Family history negative.

*History.*—He has smoked a great deal. At 18 he had a luetic infection and "secondaries," and was treated for two years. Since that time he has had occasional periods of depression. In the two months before his visit to me he had again become very much depressed and melancholy.

*Examination.*—When seen by me he had no objective symptoms of any form of nervous syphilis. A week or two later he complained that at times he had shooting pains in the legs, the right pupil was then larger than the left, but the pupils both reacted to light and to accommodation. The reflexes were present, and he had no ataxia.

*Subsequent History.*—I lost sight of him then, but saw him again six months later. He then complained that his memory was poor, and that he was unable to concentrate his mind at business. He felt generally weak. His speech was not perfectly good, his articulation being somewhat thick at times, and he now had a distinct facial tremor and Argyll-Robertson pupils. His symptoms in general were those of forgetfulness, inability to concentrate the mind, occasional headache, and depression at times, though this had improved of late. This, it seemed to me, was sufficient to justify a diagnosis of probable paresis, and what I at first considered to be merely a mild attack of melancholia seemed now to be probably a very serious condition.

*Treatment.*—He was placed on mercury and tonics and sent off to rest. He steadily improved, and at the present time, now three and a half years since I last saw him, he is perfectly well and has been attending to his work for the last three years.

*Remarks.*—This might, perhaps, be interpreted as a case of recurrent melancholia with cerebral syphilis, but I think that, at least, with the speech disturbances, Argyll-Robertson pupil and facial tremor in mind, we may assume that a paresis would naturally have developed.

Case 4.—J. R. K., aged 30; lawyer.

*History.*—He had an infection ten years before and was thoroughly treated for four years; he had had "secondaries," and during the last three years had had throat trouble, but not of a specific character. His habits are good, except that he has been a very hard worker. Two years before I saw him he began to have a slight amount of ataxia, and at the same time had some deafness. Six months ago the ataxia was very much better. One month ago he had pain in the eyes and diplopia, due to a paresis of the right externus.

*Examination.*—The fundus was normal. He had Argyll-Robertson pupils. He had great exaggeration of the patellar reflexes, no ankle clonus nor Babinski sign, some ataxia on standing and locomotion; mentally he was very nervous and excitable; he was unable to concentrate his mind and had some impairment of memory.

*Remarks.*—The diagnosis was made of impending tabo-paresis. This was two years ago. Since then he has been steadily improving and became able to resume work. He has now been attending to his work as a lawyer regularly for nearly two years, and though still ataxic and not by any means well, his mental and physical trouble at least has been improved and arrested.

Case 5.—S. B., aged 40, married; broker. Family history is good.

*History.*—He has always been a very hard-working man; he has smoked immoderately and drank moderately. He had a luetic infection five years ago, with "secondaries," which were thoroughly treated. I saw him first in October, 1903. For nearly a year previous his mental condition had been changing. His wife had noticed that he was more morose, less inclined to social life, often unreasonable, and irritable and forgetful. This was simply attributed to his overwork and to the excitement of his occupation. In August, 1903, he had an epileptiform attack during the hours of business. He recovered from this promptly and continued his work. Later, in the month of October, he had another epileptiform attack at night, followed by a period of maniacal excitement, lasting for one or two hours, in which he became quite violent.

*Examination.*—I saw him the next day. He was a large, robust man, with a very healthy physiognomy. Mentally he seemed practically normal, although he was naturally much disturbed and apprehensive over what had occurred. He exhibited to me no traces of forgetfulness or ideas of grandeur. Physically he had distinctly the Argyll-Robertson pupils, and the knee jerks were exaggerated; there was no facial tremor or hand tremor. The writing was normal. He had no lightning pains and no paresthesias.

*Subsequent History.*—This patient has now been under observation for over a year. For some time he showed an abnormal exhaustibility and irritability, of both body and mind, so that he tired very easily in walking, and had a disinclination to the mental activity involved in social intercourse. His disposition was for some time a good deal changed; he was morose, childish and suspicious of his wife, unreasonable and irritable. He, however, never showed any lack of judgment or any forgetfulness in business affairs; he was only childish in his domestic and in certain social relations. After about six months the pupils, which had been stiff, became normal the knee jerks remained about the same. He had no further convulsive seizures. At the present time this patient seems in every way to be a normal man. He had not had any severe headaches, no lightning pains, no disturbance of the spinal centers.

*Remarks.*—The probability of an impending paresis was based on the very marked syphilitic infection, which had been rather obstinate in his case, and the occurrence of two convulsions, without previous headaches, the change in his disposition and character, the forgetfulness about things in his domestic relations, a carelessness of speech and manner, which were not natural to him, and the condition of his pupils. His own physician, Dr. Sherwell of Brooklyn, and with whom I had several talks about the case, shared with me, I think, though perhaps to a less degree, the fear that he might develop paresis. This was the club held over his very impetuous nature which obliged him to change entirely his mode of life, and by reason of which I think he escaped the catastrophe.

Case 6.—H. B., aged 43, married; occupation, broker. Family history unknown.

*History.*—The patient, from boyhood, had always been a very hard-working, excitable man. He had lived a pretty fast life, drinking considerably, but he was not a smoker. He was a man whose habits of expression and thought were always very extravagant, and whose business dealings had been, normally, of a very large kind. He denied any specific infection, but I am sure he had lived a life which would have exposed him to it. In the spring of 1904 he was under a specially severe strain and had lost a good deal of money. In June he had a paresis of the left internal and right external rectus for which he consulted Dr. Koller, who kindly referred him to me.

*Examination.*—When I first saw him in July he talked in a very extravagant and excitable way, telling me remarkable stories about his business, which I believe were more extravagancies than actual delusions, and I learned from his friends that this was his way of talking. He had double vision on account of the internal rectus of the right eye. His pupils were dilated, the left larger than the right, and neither reacted to light, but reacted to accommodation; the optic nerve and vision were normal. He had a fine tremor of the hands, but none of the face or tongue, and speech seemed clear, though occasionally there was a little slip; his writing also was fairly good. The knee jerks were somewhat exaggerated; but there was no clonus. He had no ataxia and no lightning pains. The bladder and sexual functions were normal. The memory did not seem impaired.

*Treatment.*—He was given a course of hypodermic injections of mercury, and then went to Europe for six weeks, where he became more depressed. On his return he continued under the treatment of Dr. Koller for his eye condition. He gradually improved, both mentally and physically, until now his eye is nearly normal; he has no tremor, the knee jerks are less exaggerated, and his mental condition is, perhaps, better than it ever was in his life.

*Remarks.*—This case might be interpreted as possibly one of paresis, with a remission, but I feel very confident that he did not have a frank attack of paresis when he came to see me, and that he was then suffering only from a sort of grandiose excitement, with Argyll-Robertson pupils, and an eye palsy. A little further delay and he would have been fairly a paretic.

Case 7.—This case was less striking than the others, but, taken in connection with them, it has significance.

*Patient.*—J. H., aged 46; family history good; designer.

*History.*—He had syphilis at the age of 23 and was treated thoroughly by specialists. At 34 he had some mental depression and rheumatic pains. He was relieved by hypodermic treatment with mercurials. He married at 37 and had a healthy child. At 46 he had nervous attacks consisting of agitation and trembling, and he has also had a good deal of dyspepsia. On this was engrafted an "anxious depression" or mild form of hypochondriacal melancholia, which did not keep him from work. He had

no objective symptoms, but complained of his depression and dyspepsia. He gradually recovered, but a year later complained of entire loss of sexual power, annoying failure to memory, difficulty in concentration and work, and dyspeptic symptoms. This condition continued for a year, with some remissions, but finally he reported himself as practically well of everything but his sexual weakness. He had no symptoms of tabes at any time, except his sexual weakness. I watched constantly for the development of signs of paresis, but he had only the failure of memory, inability to concentrate his mind, and the initial melancholia.

The patient, from the time he was 34 till he was 46, took courses of mercury by injection. At first he took an injection every two weeks for two years. They acted on him as a tonic, the effect lasting for a week or more. Later he took the injections at much longer intervals.

These histories, I might repeat, may seem to present inconclusive evidence of the real existence of paresis. I think, however, that most of those who are familiar with such cases will admit that they have seen paresis beginning in this way and going on to the full-fledged condition. Such cases certainly have occurred in my experience, and usually after these initial symptoms were already matters of the previous history.

The importance of recognizing paresis before it is in a way real paresis is the very evident lesson of these histories.

*Treatment.*—In conclusion, I would say a word only in regard to the treatment of these conditions. It seems to me that if the cases are recognized early there is no need of any very novel methods of treatment; there were none employed here. I believe that the patients should at once be turned entirely from their former modes of life; that they should be sent where they can get rest and fresh air; that they should receive, if possible, hypodermics of the bichlorid or salicylate of mercury, and that this should be accompanied or followed with iodid of potassium and tonic measures. I attach special importance to the effect of hypodermic medication, though all the patients did not receive it. It is not always necessary to give large doses, i. e., gr. $\frac{1}{4}$ twice a week is sometimes enough, but this may need to be kept up for two or three months. In other cases gr. ii or even gr. iii once or twice a week are required. The technic requires care. During the course of treatment there should be a very liberal use of lukewarm and hot bathing (a warm bath every day and a hot bath once or more weekly), and every possible attention should be given to the general nutrition of the patient.

With these measures I believe that a good proportion of persons who are threatened with paresis can be permanently helped, and it is my hope that the medical profession will become trained to recognize these cases so quickly that before many years we may get the same gratifying results in paresis that we do in tabes.

I have to express my obligations to Dr. Carl Koller, Dr. Alex. Duane, Dr. Samuel Sherwell, Dr. Jas. R. King, of Watkins, and Dr. Sollace Mitchell, of Jacksonville, Fla., for referring some of the cases to me, for aid in treatment and for furnishing me special notes for the histories.

SECOND PAPER, READ NOVEMBER 3, 1909

In a paper read before this society five years ago, I made a contention that it was possible to arrest, and probably to arrest permanently, certain cases of paresis when they were observed in the earliest stages and submitted to prompt and prolonged treatment. To the objection that the cases cited in illustration of my point were not really cases of paresis, but of cerebral syphilis or "pseudoparesis," I could answer only by contending that there were no possible criteria by which we could distinguish "pseudoparesis" and certain forms of cerebral syphilis from paresis itself, except by saying that when the patients recovered the cases were false, and when the patients died the cases were real. In the pseudoparetic cases as in true cases, we have headaches, convulsions, syncopal and aphasic attacks, disturbance of the pupillary reflexes, tremors, cranial nerve palsies, even attacks of hemiplegia, lymphocytosis of the cerebrospinal fluid, and positive reaction to Wassermann's test. In each kind of case also, we find changes in character, increased irritability, inability to conduct business affairs, defects of attention, somnolence, confusion and quick exhaustibility of the mental processes on exertion. In cases in which paresis is firmly established, with grandiose delusions, real dementia, marked facial and tongue tremors, speech defects, involvement of the pyramidal tracts, I make no claim that the disease can receive, at the most, more than a remission.

The clinical facts on which my contention is based have been so abundant and positive since I first wrote on this subject that it seemed almost unnecessary to emphasize the point further. I am led to do so, however, because my view has been misunderstood by some who have jumped to the conclusion that I have

asserted that paresis can be cured when fully developed; also, because my view as to the essential unity of nervous syphilis and parasyphilis has been so strongly confirmed by the discovery of the pale spirochete and the evidence of its activity by Wassermann and other tests in parasyphilitic conditions.

The observations of Drs. Noguchi and Moore[1] show that in general paresis and in cerebral and nervous syphilis, there is a positive reaction both to the butyric acid and the Wassermann test; also that there is a practically equal evidence of lymphocytosis in the cerebrospinal fluids. Similar results were obtained by Dr. Rossanoff[2] in his observations recently presented before the New York Academy of Medicine. Dr. Kaplan's observations presented at the same time lead to the same conclusions. It is very well established, then, that so far as the blood and the cerebrospinal fluid tests go, there is no qualitative difference in the reactions between paresis and so-called syphilis of the nervous system. There is indeed a rather stronger and more positive reaction in paresis than in the ordinary syphilis of the nervous system.[3]

[1] Noguchi, H., and Moore, J. W. The Butyric Acid Test for Syphilis in the Diagnosis of Metasyphilitic and Other Nervous Disorders, Jour. Exper. Med., July 17, 1909, abstr. in The Journal A. M. A., Aug. 14, 1909, LIII, 591.

[2] One of Dr. Rossanoff's conclusions is the following: "Inasmuch as the Wassermann reaction and the butyric acid reactions seem to indicate syphilis only when it exists in an active or potentially active form, their regular occurrence in general paresis would tend to prove that that disease is a manifestation of active syphilis of activity of the *Spirochæta pallida*. While the evidence for this view is not as yet complete, it is sufficient to justify its being used as a basis of therapeutic essay." Rossanoff also cites a case which was diagnosticated general paresis. The patient, however, recovered under treatment by mercuric injections, whereupon the diagnosis was changed to cerebral syphilis. While it cannot be proved, clinical experience in other cases justifies one in saying that if this patient had not received treatment he would have died with the symptoms of paresis. Cases like this have occurred in my experience and have been potent in forcing me to my conclusions as to the nature and management of this disease.

[3] Apelt's later observations (Arch. f. Psychiat., XLVI, No. 1) only show that the "Phase I" or globulin reaction does not occur so often in so-called cerebral lues or luetic neurasthenia as it does in tabes and paresis, but it does occur, and all the serum and blood tests indicate only a quantitative, not a qualitative, difference between the conditions.

The attempt to find a test which should distinguish between paresis and nervous syphilis or "syphilitic neurasthenia," has been made by Nonne and some of his followers. The most recent observations regarding the utility of what is known as the Phase I Test of Nonne failed to show that it is of any distinct value in distinguishing between these two conditions (Noguchi and Moore[1]). It follows, then, that since there is an essential underlying unity between true paresis and "luetic neurasthenia," "pseudoparesis," "nervous syphilis," there is no reason to suppose that if we can cure one we can not also forestall and even cure the other. This is what I assert to be possible and also, that it is not infrequently done. Over and over again, it has been my experience to see luetic patients suffering in almost the same way, nervously and mentally, and yet, in one case, after a time, a true paresis develops, and in the other case it does not. And, furthermore, it has been my experience to see patients who have, clinically, developed nearly all the signs of paresis, improved, and remained practically well for years.[4]

The ordinary, and perhaps I should say asylum view of prognosis of paresis, has been recently presented by Dr. Green,[5] who made a very careful study of 200 cases, with reference to this point. He puts the limit of the duration of the disease at five years, this occurring only in the young; and he does not speak even of the possibility of cures, or long remissions. Such a view expresses, I think, a narrow and misleading conception of the malady, for paresis exists sometimes for years before it reaches the alienist or the hospital. A writer in one of the medical weeklies, during

[4] Sometimes cases which present mainly the appearance of cerebral syphilis do not improve under treatment, but go on steadily to a fatal termination with all the symptoms of paresis. Two patients came under my care at about the same time, beginning in the same way. One is now apparently in active health; the other died of paretic dementia. There must, then, be some plus factor in the matter which is potent to bring about the unfavorable result. This may be inadequate or belated treatment, or it may be a more degenerate, unstable brain. The luetic poison quickly brings about hopeless toxic changes in ablotrophic cells. Paresis that is fatal, then, is that in which the luetic infection is severe and uncontrollable, or that in which the brain-cell, being congenitally weak, easily falls prey to the toxin. This is the only difference which I can see between cerebral syphilis and parasyphilis."

[5] Green, Jour. Ment. Sc., 1908 or 1909.

the past summer, also states unequivocally that paresis is not only never cured, but surely and always kills, i. e., it is not even permanently arrested. This also expresses what I should term a very juvenile view of paresis. Quite appropriately this writer quotes a French novelist in support of his position, but does not, I think, get any appreciation of some more serious writers. As against Guy de Maupassant, I would quote Alzheimer, who states that he has most positive evidence that paresis has remained unprogressive for many years.

I wish now to put on record the final history of the cases which I reported five years ago, as an illustration of arrest in preparetic states. The details of the early history of the cases are given in the article referred to.[1] I shall add a few further reports of cases illustrating the clinical fact which I am trying to prove, for I know that it is only by the presentation of such data, accurately and impartially given, that any conviction can be produced; the minds of many alienists, at least, seem to be absolutely fixed in an attitude of pessimism towards the prognosis of paresis in any of its stages. The cases are referred to in the order they were reported originally.

Case 1.—A broker, aged 40, had an attack of melancholia, with the somatic symptoms indicating paresis; that diagnosis was made in his case. He had tremors and Argyll-Robertson pupils, but he had shown no dementia. He improved in the sanitarium and kept well for three years afterward, at which time I reported the case. He continued well for four years more, but he lived a life of great self-indulgence. He had losses in business, and finally developed genuine symptoms of paresis and died in about a year. He had had, therefore, a remission of seven years, during which he was in every respect a normal man.

Case 2.—The patient had continued well for four years from the time when I saw him, when his symptoms were marked. He had had a luetic infection twelve years before. His symptoms were those of convulsions, speech disturbance, feeble memory and depression. He has continued well, and is now in active business. It is eight years since he recovered.

Case 3.—A man, aged 34, had a speech disturbance, Argyll-Robertson pupils, facial tremor and depression to the point of melancholia. When I reported his case he had been attending to his work and was well for the last three years. I saw him again in the past year and he had continued well, so that his remission has thus far lasted eight years.

Case 4.—A lawyer, aged 30, had all the evidences of a beginning taboparesis. The progress of his disease was arrested, and for seven years he has been able to do his professional work with a fair degree of success.

Case 5.—A broker, aged 40, had had an infection. His trouble began with a violent epileptic attack, followed by periods of maniacal excitement. For a year he had a great deal of mental irritability and depression, and a certain degree of weakness of comprehension and judgment. He has now continued well for six years since the first attack.

Case 6.—A man, aged 43, a broker, lost all his money in Wall Street, and then tried to steal some to make good. He developed a melancholia and killed himself.

Case 7.—This was one of those types of luetic infection, followed by anxious depression or a mild form of hypochondriacal melancholia. For twelve years the patient used to have an injection of mercury, every two weeks. It is now nine years since he was under systematic treatment and he continues well.

The following further cases illustrate the preparetic state and its possible outcome:

Case 8.—T. S., aged 46, lawyer, came to me in October, 1906. There was no hereditary history of importance. His father lived to 70, his mother died at 60, of an accident. The patient was married and had three healthy children. He had contracted lues about twenty-three years before coming under my observation. He suffered severely and, though he was treated, his health broke down and he had to rest from business. After his recovery, i. e., for about twenty years, he had been quite well and had seen nothing of doctors until his present illness. About three and one-half years before I first saw him he suffered from sexual weakness, which had gradually progressed till he had become practically impotent. Of late his bladder had been a little weak. Two years before coming under my care he had lost consciousness suddenly and was aphasic for two days. His capacity to speak in court had been lessened since then. One year before the time when I saw him he had an attack of severe vertigo. Since then his mental powers had been lessened He could not work long, could not concentrate, was forgetful, and had attacks of vertigo. He was very somnolent, sleeping ten hours at night and in the afternoon. He was depressed, emotional and unable to do much work of any kind. His gait and stature were normal, speech clear, handwriting much impaired. The knee jerks and ankle jerks were present, but uneven. The pupils were uneven and did not react to light, but did to accommodation. The left pupil was irregular in shape. The patient had no anesthesias, lightning pains or ataxia. There was tremor of the hands, and the face showed distinct facial tremor. The main mental symptoms were incapacity to work, mental inadequacy, defective memory, emotional excitability (irritability and depression) and somnolence. The physical symptoms were changes in deep reflexes, Argyll-Robertson pupils, facial tremor, defects of handwriting, sexual impotence without local cause, as attested by Dr. Eugene Fuller, who saw him in consultation, and

vesical weakness. It was six months before the patient improved very much, but at the end of that time he was very much better, except of the sexual impotence, and the pupils. His memory was good, his somnolence and apathy better. A letter received from him recently states that he is well, nearly three and one half years since I first saw him.

Such a case as this is illustrative of a type which I have also seen progress till all the marks of paresis are developed.

Case 9.—J. E., aged 43, single, occupation engineer. The history furnished by his family physician, Dr. Parslow, is as follows: The patient's father died of Bright's disease, at 69; the mother was living in good health; he had four sisters, all living. One had epilepsy for some years, but recovered. Three aunts died of tuberculosis.

The patient was always robust; used alcohol and tobacco moderately. His habits were excellent, with the exception of sexual excess, which undoubtedly made serious inroads on the patient's vitality. About three weeks after connection, in the latter part of 1901, three hard chancres appeared on the dorsum of the penis, followed by adenitis, roseola, etc. The secondary stage ran a mild course. Mercurial treatment was faithfully carried out. About one year after the primary sore, the patient was stricken with left hemiplegia. At this time, a neurologist saw him. Mercury by inunction was pushed. The motion of the left arm was seriously impaired, but improved. After the patient was up and around, it was noticed that he would fall asleep at his work, especially after eating; he was very hard to arouse mornings, even after ten or twelve hours' sleep. As he was in charge of complicated machinery, his employers had to displace him. His friends noticed that he acted queerly at times. Dr. Parslow never noticed any delusions or hallucinations. The patient wept easily, and his mental depression was marked; his countenance changed from a bright and intelligent to a dull and stupid expression. He was given iodids in large doses, but failed to improve.

When examined by me, he still showed some evidences of the left hemiplegia, the arm being somewhat contractured, and there were slight athetoid movements in the left hand. There was no anesthesia or ataxia. The reflexes were exaggerated, more on the left than on the right, but there was no clonus. The pupils were sluggish, but not actually of the Argyll-Robertson type. Mentally, he was simply very slow and forgetful; did not have even the ordinary school knowledge, nor could he recall the ordinary events of the day. He would fall asleep during the daytime and sleep all through the night. He had no cranial nerve palsies; no speech defects, but had a little tremor of the face and hands. He did not show any actual neglect of the person, and his sphincters were intact. He could read a little and write imperfectly. His condition had been gradually getting worse, notwithstanding large doses of the iodid of potassium, amounting to 120 grams a day.

The patient was put on hypodermic injections of salicylate of mercury, and gradually began to improve, and in about six months time he seemed well, and went back to his work.

I saw him a year ago, five years after my first visit. He was then well mentally in every way. His speech was good, his memory clear, and he simply had some relics of his old hemiplegia.

This is a case of cerebral syphilis, but it was passing into dementia. The patient already had tremor, sluggish pupils, exaggerated reflexes. He was dull, slow, had lost his knowledge of things, was extremely forgetful, queer in actions, depressed, emotional, somnolent and partially demented.

CASE 10.—E. A. M., male, aged 52, married, with two healthy children, came to see me first in May, 1906, suffering from tabetic pains. He had had a luetic infection at the age of 20, and was thoroughly treated for two years; he had had no luetic manifestations since then. He had been a hard worker at the dry-goods business. His habits were good; he used no alcohol or tobacco in excess. In 1898 he broke down with what was called "nervous prostration," and was unable to work for about six months. Two years later, in 1900, he began to have the characteristic pains of tabes, and had been treated for these symptoms, and these alone, since that time.

Status in 1906: The gait was normal; there was slight Brauch-Romberg sign present, slight ataxia of the hands to the finger-nose test. Argyll-Robertson pupils. unequal in size, knee jerks and ankle jerks absent, tremor of the facial muscles and of the hands, no hypotonia, no anesthesia, bladder slow, sexual function very weak, tachycardia, but no valvular lesion. Mentally, the patient was irritable and quite forgetful; he had a slightly exaggerated manner, was a little *exalté*, was easily confused, could not play cards without making mistakes, could not sign his name on account of nervousness and tremor. He was somnolent during the day, falling asleep at his desk, was not able to read except for a few moments, was unable to do his work well, becoming easily confused and exhausted. There was a general change in his character, so marked that his wife came to consult me about it. He had no delusions, but was somewhat depressed.

The diagnosis, in 1906, was tabes and developing tabo-paresis. November, 1909, after several months rest and treatment, part of the time at a sanitarium, the patient returned to business and he has continued at it ever since. I have seen him at intervals during the past three and a half years. He is now mentally normal; he is no longer forgetful and can attend to his work. He can write and dictate readily. Facial tremor is gone. The tabetic symptoms are the same, almost exactly, as they were three years ago.

CASE 11.—W. D., aged 30, married, denies infection. In 1902 he gradually developed a spastic paraplegia with bladder and sexual weakness, the disease taking the course of Erb's syphilitic spinal paralysis. In the fall the patient developed delusions of grandeur, and when examined by me he had all the somatic and mental symptoms of general paresis, with facial tremors, Argyll-Robertson pupils, and involvement of the pyramidal tracts. This diagnosis was confirmed by Dr. Frederick Peterson and the physician in charge of the institution to which the patient was committed. He was insane and delusional for nearly a year, but gradually improved, and has now been practically well of his mental trouble for over seven years. He has not known quite the mental vigor of his former years, and his spinal symptoms have remained about the same. The total duration of the condition is now nearly eight years.

This is a remission in general paresis, and it is not one of the class of cases on which I base my remarks. It is admitted by all that there may be long remissions in general paresis.

I could cite other cases from my notes, but I prefer to get the evidence of other neurologists, and Dr. Joseph Collins has kindly placed the two following cases at my disposal:

CASE 12.—A man, aged 45, stock broker, unmarried, was seen May 31, 1904. He had had syphilis when 23, an apparently moderate infection, treated by a prominent specialist for three years and pronounced cured. When 31 years old the patient had an attack of diphtheria; when 38, an attack of typhoid fever.

The first symptoms of the present disorder, of which the patient is cognizant, occurred in 1903. He was in bathing. The propinquity of a certain person was responsible for extreme erethism, followed after a long time by orgasm. The patient experienced a profoundly disagreeable sensation in the head, and had a feeling of confusion and syncope. Following this, he was in bed for a fortnight, and his physician who treated him said that he had a heat-stroke. After that time the patient was subject to periods of profound depression. He lacked initiative, was moody, sullen, unsociable, and impotent. He frequently made mistakes in his orders and thought he had to give up business. Depression was his worst symptom. The pupils were unequal; one knee jerk was absent, and there was distinct slowing of speech. This man took an enormous amount of mercury, by hypodermic injections and inunctions, and is today practically well. The physical signs remain the same, save that the speech has become distinct. He is able to transact his business as well as ever.

CASE 13.—A man, aged 46, had syphilis when 21; he was rather alcoholic. When 38 years old he developed symptoms of mental confusion, associated at times with violence, but depression was his chief

symptom. Gradually he became indifferent to his family, exhibited loss of self-control and was most easily excited and annoyed. Examination showed pupils that were unresponsive to light, slight tremor of the lips, tongue and hands, and exaggerated knee jerks. He seemed to have insight into his condition and explained his inability to work by saying that he was weak. He would frequently make mistakes in writing, however, and displayed evidences of marked forgetfulness. He was put under intensive mercurial treatment, and today, six years after I first saw him, he is running a store.

The question in all these cases, of course, comes up whether these patients really were suffering from paresis in an extremely early stage. Some authorities seem to take the position that we can never be sure we have a case of paresis until the patient is dead, and the characteristic anatomic changes are found in the cortex of the brain. This anatomic test of the existence of paresis is an unworkable one, and would make the talents and acuteness of the clinician practically of no value. Besides, it seems to me to be utterly without foundation. I do not believe that in the very early stages of paresis any anatomic change exists which could be detected by the microscope, and if we have to wait for our diagnosis of the disease until these anatomic changes occur, I can well agree that the prognosis must always be hopeless. But it is my conviction that we can recognize the malady before any appreciable changes do occur, or at least, changes which are beyond hope of repair or arrest. Let me illustrate this position by the following history:

Case 14.—A young man, a lawyer, aged 33, was infected with syphilis about eleven years before he was seen by me. He was treated for the disease continuously for two years, and after that period, every six months, for about six years. He continued his professional work, and was very successful in it, until about a year before I saw him, when he suffered from symptoms of what was thought to be neurasthenia. He was treated on this basis and went out West, took active exercise on horseback. He improved somewhat, but then became considerably worse, and was finally brought to me in January, 1909, when he had distinctly the symptoms of paresis—forgetfulness, inability to concentrate, confusion of ideas, irritability, a good deal of mental depression, and certain almost delusional ideas regarding the way in which he was treated by his friends. Physically, he had distinct and characteristic dysarthria, tremors of the face and hands, exaggerated reflexes, and Argyll-Robertson pupils. The Wassermann test was positive. He improved a little for a time, but at

the end of the following summer he was distinctly demented and delusional, and had all the characteristic physical symptoms of general paresis. These progressed, and he is now in the third stage of the malady.

The point of interest in this case is this: On inquiring carefully about the patient's earlier years, I found that five years before I had seen him, he had had an attack of mental depression, of a somewhat hypochondriacal type, such as so frequently ushers in paresis. This had occurred without special cause, and in a man whose habits were good, and who had inherited a sound constitution. I further learned that after he recovered from this depression he had been a little different in temperament and character, a little more irritable and unreasonable, a little more egotistical and expansive than usual, his friends thinking that he was simply rather more conceited and difficult to get on with than before. But, in the light of subsequent events, it seems clear that these were the mental forerunners of his later attack, because when the later attack came on, all these characteristics were simply very greatly exaggerated. Thus, it seems that this attack of paresis can be traced back to a period of five years, during which time the patient achieved a really brilliant success in his profession, transacting his work effectively, and not suffering from any physical troubles whatever. It seems incredible that during all these years of successful professional work and social activity, he had any serious degenerative changes in the cortex of the brain, which changes are supposed to be necessary to enable one to make a diagnosis of paresis.

If the history of cases of paresis could be traced back in all instances, to the very remotest beginning, I believe that in a good many instances this prolonged preliminary period of slight mental perversion might be discovered, and in this case I think that if treatment had been undertaken along proper lines three years before, or two years before, the malady might have been kept in check.

The onset of a parasyphilis, occurring in persons who have had an infection, takes place in different ways as follows:

1. Acute symptoms of syphilitic exudates in the brain, ending promptly or later in paresis, or ending in cure, with or without mental symptoms, or ending in some deterioration with final serious cerebral vascular changes.

2. Acute mental symptoms, maniac or melancholic, ending in cure or paresis.

3. Tabetic and paretic symptoms, ending in taboparesis or in tabes with arrest of paresis.

4. Insidious mental and physical deterioration, ending in paresis.

The above initial conditions all may or may not end in paresis, depending on treatment, the constitution of the patient, and the intensity of the infection. Often they go on until nearly every symptom of paresis appears, yet they are even then arrested. The clinical phenomena, from the beginning to near the end, are produced by the same pathologic agent, and often can be controlled by treatment, even when the signs are ominously identical with early symptoms of general paralysis. This is what I mean by "cure of early paresis."

# THE DIAGNOSIS OF GENERAL PARALYSIS[1]

By C. Macfie Campbell, M.D.

ASSISTANT PHYSICIAN BLOOMINGDALE HOSPITAL, NEW YORK

The differential diagnosis of general paralysis is an extremely wide subject in view of the polymorphous character of the disease. Not only do various forms of organic brain disease present clinical pictures difficult to distinguish from general paralysis, but the latter may first manifest itself in a form difficult to distinguish from that of one of the so-called functional psychoses; thus a manic attack may be the first expression of a general paralysis.

The question of the early diagnosis of general paralysis, and the discussion of those symptoms accompanying a so-called functional symptom-complex which enable one to diagnose the paralytic process underlying it, will no doubt be taken up by others.

I propose to contribute to the discussion several cases where the difficulty of diagnosis was not merely transitory, nor due to the disease being in an incipient stage; but where in a well-advanced stage of the disease it was extremely difficult to know how much weight to lay upon the various symptoms, and to come to a conclusion as to the process at the bottom of the disease.

It will be necessary in regard to some of these cases to refer to the result of the anatomical examination which decided the diagnosis; before doing so, and in order not to beg the question, one must first be clear as to the present state of our knowledge of the histopathology of general paralysis.

The most satisfactory and most recent statement on that topic is to be found in the first volume of Nissl's publications from the laboratory of the Heidelberg clinic. The volume contains the results of the work of Alzheimer and of Nissl; both, working at the same question upon independent material, came to the same conclusion, that in general paralysis there are always characteristic histopathological changes which enable a positive diagnosis to be made. A slight reservation must be made with regard to certain cases of idiocy upon the basis of a non-purulent encephalitis, with

[1] Read before the New York Psychiatrical Society, November 7, 1906.

regard to which there is not as yet sufficient material for definite statements to be made. In the work referred to, Nissl discusses in great detail certain morphological questions, and emphasizes the wide biological and pathological issues, while Alzheimer limits himself more strictly to the immediate needs of histopathological and clinical differentiation. Reference, therefore, will be made chiefly to the work of Alzheimer.

The material which Alzheimer used consisted of 170 consecutive cases of clinically undoubted general paralysis; in every case he found certain histological changes in the cortex; he concluded, therefore, that he had a definite anatomical criterion to apply to all cases of doubtful clinical diagnosis.

This anatomical criterion is not claimed as forming the essence of the paralytic process; the same cortical changes were found by Nissl in a dog and two rabbits; they form, however, a trustworthy empirical criterion, which every case claimed as general paralysis must satisfy.

Macroscopic examination is not sufficient to settle conclusively a disputed case; at the autopsy the brain of a general paralytic may show no macroscopic evidence of general paralysis; on the other hand Alzheimer refers to a case diagnosed in life as senile dementia, which presented at the autopsy thickening of the calvarium with disappearance of the diploë, thickening and opacity of the pia with pial collections of fluid, hydrocephalus internus and externus, ependymal granulations, marked atrophy of the brain, especially in the frontal region; microscopical examination, however, confirmed the clinical diagnosis of senile dementia. Such a case demonstrates the necessity of microscopical examination.

The following is a brief summary of the histological changes characteristic of general paralysis.

1. In every case the pia mater shows diffuse changes, usually most marked over the frontal lobe; these changes consist essentially of an infiltration of the pia with cellular elements, plasma-cells, lymphocytes and mast-cells; in addition the vessel-walls show progressive and regressive changes.

With regard to the cortex in general paralysis it is convenient to discuss first the mesodermal elements and then the ectodermal elements.

2. In every case there is proliferation of the endothelial cells of the vessels with a marked tendency to the new formation of vessels through sprouting and vascularization of the proliferated intima. There is increase of the elastica and proliferation of the adventitia. There is widening and infiltration of the lymph spaces, which exist in the adventitial coat of the vessel wall. Among the infiltrating cells, plasma-cells are the most numerous: they are never absent in a case of general paralysis, even in the most acute. Lymphocytes and mast-cells may also be found in the infiltrate. In advanced cases the vessel walls show regressive changes. Regularly in general paralysis, we find long rod-shaped or sausage-shaped cells in the cortex, their long diameter tending to run parallel with the medullary rays.

As to the ectodermal elements:

3. The nerve-cells show a great variety of degenerative forms, the meaning of which is as yet quite obscure; in advanced cases the nerve cells have in part disappeared. The usual orderly arrangement of the cells in the cortex is more or less disturbed. There is usually a considerable degeneration of the medullated fibers in the cortex.

With regard to the non-nervous ectodermal tissue, i. e., the neuroglia:

4. There is always a marked proliferation of the glia; this proliferation leads at first to the formation of numerous large glia cells which form a large number of fibers, and, in very advanced cases, dense tissue of thick glia fibers. The most marked increase is situated in the molecular layer and along the vessel sheaths.

The changes in the rest of the nervous system in general paralysis, the nature and degree of affection of the central ganglia, cerebellum, cord, etc., need not at present be discussed.

Lissauer has described cases of atypical general paralysis, in which focal symptoms precede the symptoms of mental disorder; in these cases one frequently has from time to time apoplectiform attacks, each attack leaving the same residual defect—hemiparesis, hemiplegia, aphasia—which is frequently transitory.

In such cases the typical cortical changes of general paralysis are found, the focal symptoms not being due to the addition of syphilitic or arteriosclerotic or other lesions, but to the unusual severity of the process in certain areas, the central convolutions,

the temporal lobe, etc. In other cases, where the distribution of the process has at first been the usual one, later in the disease the changes may become most severe in the posterior half of the cortex and focal symptoms may then develop. Transition forms between the typical and the atypical general paralysis are numerous.

In certain cases we find general paralysis associated with tabes, and that may occur in two ways: tabetic changes may develop during the course of general paralysis, or a person, who has for long been a tabetic, may later show signs of general paralysis. The tabes of a general paralytic is histologically little different from that of a non-paralytic; different systems in the posterior columns, however, tend to be first affected and a slight cellular exudate is frequently seen in the posterior columns of the general paralytic with tabes, but has not been observed by Alzheimer in tabes itself.

In general paralysis, there is a constant histopathological picture. Can one always distinguish this from the picture found in cases of alcoholism, senile dementia, arteriosclerotic brain disorder and brain syphilis? In other words, in doubtful clinical cases, will microscopical examination enable one to differentiate between these groups?

With regard to the first three groups there is little difficulty, and it is not necessary to go over the differences in the degenerative changes found in each; a diffuse perivascular infiltrate with plasma cells is not found in any of these conditions.

The differential diagnosis between general paralysis and brain syphilis is more difficult, but is, however, always possible; at least, one can always say whether the case is general paralysis or not; it is not always easy to determine whether, in addition, there is a syphilitic element present. Brain syphilis is a general term which includes gumma of the brain, syphilitic endarteritis of the cerebral vessels, either of the large vessels (Heubner's form), or of the terminal vessels (Nissl's non-inflammatory form of brain syphilis), and finally syphilitic meningo-encephalitis, with or without gummatous formation. Each of these presents a definite histopathological picture and can be separated from general paralysis. The most difficult cases are those belonging to the last group—i. e., syphilitic meningo-encephalitis; careful microscopical examination enables one here, too, to separate the two conditions. The pictures in

Alzheimer's work illustrate well the two conditions. The meningo-encephalitis shows the most marked infiltration in the pia, which, along with the vessel walls and the cranial nerves passing through the membranes, is infiltrated with lymphocytes. The process spreads from the pia into the cortex, affecting the superficial layers more than the deeper, sometimes obliterating the border between pia and cortex. Where the affection of the pia is localized the cortical changes show a similarly circumscribed distribution. The condition may be complicated by gummatous nodes, and by softening due to thrombosis.

Such a picture is quite different from that of general paralysis where the severity of the cortical change does not necessarily correspond with that of the meningeal change in the same neighborhood, and where the cortex shows parenchymatous degeneration and vascular changes diffusely distributed throughout the whole cortex. The behavior of the cells of the infiltrate is different in the two conditions. In the syphilitic meningitis the infiltrate, composed chiefly of lymphocytes, is not arrested by the limits of the vessel walls nor of the nerves which pass through the pia, it penetrates both. The cell changes in the pia do not show the variety of progressive and regressive forms found in general paralysis.

A syphilitic meningo-encephalitis of the convexity is almost always accompanied by a syphilitic meningo-myelitis.

Mahaim says that it is impossible to differentiate general paralysis from diffuse brain syphilis; but the form which causes most difficulty is that already discussed and the individuality of which has been established. His views are based upon an insufficient study of the infiltrate in the various conditions.

Klippel considers general paralysis a clinical syndrome which may arise on an alcoholic, syphilitic, arteriosclerotic or other basis. He bases this statement upon quite erroneous ideas, the criticism of which would necessitate a somewhat detailed statement of his position. As a matter of fact, the present state of our knowledge of pathological anatomy has enabled us to distinctly separate the various processes, arteriosclerotic, senile, alcoholic, syphilitic and general paralytic; if then, clinically, one is not always able to distinguish between these groups, that is no reason for refusing to recognize them as different conditions; it

is simply a reason to push further our clinical analysis. The clinical differentiation of these groups is not so far forward as the anatomical differentiation. There is an urgent necessity for well-analyzed clinical material, so that we can make full use of the assistance which microscopical examination offers us. From this point of view the following cases are presented.

Case 1.—William C.; 47 years of age, admitted to Manhattan State Hospital on August 25, 1905; died May 3, 1906.

His father was alcoholic; he himself was a neurotic child, had night terrors, walked and talked in his sleep. He developed normally, later became addicted to alcohol, married in 1891, having had a chancre six months previously, for which he received only three months' treatment. He infected his wife, and she had a disastrous series of pregnancies, but finally a living and healthy child. Ever since his marriage, the patient drank immoderately, indulged in excessive sexual intercourse, tormented his wife with his morbid jealousy and suspicions, so that she left him more than once. As far back as 1897, he would suspect poison, and imagine that his relatives and associates dealt unfairly with him; his waking thought was apparently considerably influenced by dreams.

Focal symptoms first came on in 1900. In 1900, towards the end of summer, his wife noticed squint of the right eye; this persisted till November. In September of the same year, he had an attack of dysarthria and staggering; no definite weakness of the limbs of either side was noticed. He recovered after two months in bed, his speech returning apparently to its normal condition. He was able to resume work.

1901. Again fatigued and without appetite; he lost the power of his right leg "from the knee downwards," the weakness not being abrupt in onset but coming on in the course of a few days; speech was thick.

1901–1903. Unable to work; he dragged the right leg; two or three sudden attacks of paralysis of the right arm of one half day's duration, with weakness of the right side for about a week.

1903. One day he suddenly fell; when seen one hour later, he was a little excited, able to speak, his *left* arm suddenly became paralyzed, he could not speak for one hour; he was then able to walk, went to work the next day.

1903–1905. Worked as night watchman, gradually became weaker, did not recognize that he was an invalid, made light of his hemiplegia.

1904. Occasional difficulty in controlling his urine.

1905. July. One day at noon he was found unconscious; half an hour later he was excited, but unable to speak, he made signs, then said "that's better." For one week he was semi-conscious, disoriented, didn't know his wife; he remained at home for five weeks, was disoriented, had hallucinations of sight—"didn't you see them men?"—thought medicine was poison. In Bellevue Hospital he thought he was in St. Stephen's Church, had no idea of why he was there.

On admission to M. S. H. he was at first excited and pugnacious, but on examination quiet and agreeable, anxious to go home; he was able to maintain a conversation. He was happy, felt first-rate, did not resent being with crazy people; "this is Manhattan Life Insurance-Bellevue Hospital," he gave the date correctly. He had fair memory of remote and recent events, but his dates showed discrepancies, e. g., "this is 1906 —born in 1862—47 years old." Dilapidation of general knowledge; poor retention; no insight.

August–October. Mildly demented, whimpering easily over his detention.

October 25th. Ptosis of the left eye developed, became complete in a week, cleared up after four weeks.

*Physical Status.*—October 25, 1905. There was weakness of the right face, arm and leg. Knee-jerks and Achilles-jerks exaggerated, especially the right. The gait was rather unsteady as well as having the "mowing" character. Babinski reflex on both sides. Slight ptosis of the left eye; left pupil dilated, did not react to light nor on accommodation; right pupil smaller, reacted sluggishly to light. Fundi normal. Speech: slurring, slightly sticking, without omissions or transpositions, but with occasional insertion of r. Writing: tremulous with omissions and distortions, e. g., "meth-espical" (methodist episcopal), "bittililery" (artillery). There was no gross sensory disorder. Lymphocytosis of the cerebrospinal fluid.

During his stay in hospital, the patient continued to present this picture of mild general dilapidation, with no adequate realization of the mental and physical impairment, but showing no definite expansiveness nor other abnormal mental trend while in hospital.

On April 30, 1906, the patient had a general convulsion, became unconscious; he died on May 3.

The question whether one had a right in this case to consider a diagnosis of general paralysis as established was discussed during the lifetime of the patient. Our present knowledge of the symptoms of brain syphilis is inadequate and based on a very limited material, and it was felt that to exclude the diagnosis of brain syphilis altogether was to assume a knowledge of the same which we are far from possessing.

The possibility of brain syphilis was suggested by the variety of the neurological symptoms—squint in 1900, a series of right-sided attacks with dysarthria from 1901 to 1903, transitory paralysis of the *left* arm in 1903 (if the wife's statement can be relied on), the apoplectiform attack preceding commitment, the transitory ptosis of the left eye which developed in hospital. Such a train of symptoms seemed in keeping with what we know of the course

of brain syphilis, and it was considered possible that the underlying anatomical process might be a syphilitic meningo-encephalitis with syphilitic endarteritis, the hemiplegic attacks being due to the vascular changes; the mild dementia might then be regarded as an ordinary post-apoplectic dementia.

This hypothesis, however, did not seem to explain various features in the case. The want of insight into his mental and physical impairment, and his general optimism seemed to be more than is usual in a non-paralytic dementia; it is true that in some cases of the latter a similar attitude may be seen, and the difference in degree is difficult to estimate. Repeated tests of his memory showed a striking difficulty in handling dates, and an inability to correct discrepancies; e. g.: Age? "48." Present year? "1906, then I must be 58." Are you 58? "No—that would be 1858—I would be 50 then—this is only 6—that would be right, 58 years of age." 58 years of age? "Yes, sir." Are you 50 years of age? "No, I would be 48—I made a mistake—48 and 58—1858, and then 50—40 more—would be 1906,—wouldn't it—18 and 46 more." At the same time the patient showed great pertinacity in trying to correct discrepancies.

The speech also (e. g., bittililery) seemed to show a more profound defect in the grasp of words than is usually met with in brain syphilis.

The physical symptom-complex seemed consistent with either view of the case, although the variety of the neurological symptoms pointed rather toward brain syphilis.

On the basis of the above analysis, the conclusion was that the patient was probably a case of general paralysis, but that, in so far as we are not entitled to deny the possibility of the presence of the above mental defects in brain syphilis, and in view of the neurological course, the diagnosis of general paralysis was not considered as established.

At the autopsy there were noted slight atrophy of the frontal region, rather more marked on the left side, slight general thickening of the pia, granulations in the fourth ventricle. In the knee of the right internal capsule there was an old focus of softening, and another in the left side of the hind brain, involving the pyramidal fibers. There was a gumma in the left centrum semiovale, connected with the pia of the insula, another in the right parieto-

occipital fissure. Histological examination showed a syphilitic meningitis, of varying grade, with slight extension into the cortex.

The case, therefore, was one of cerebral syphilis.

CASE 2.—J. L. W.; 43; admitted to M. S. H. August 4, 1906; died August 26, 1906.

The patient was a journalist, who, in 1898, at the age of 35, had a chancre for which he was treated for 4–6 months. In the summer of 1901 he had diplopia, which improved with two months' treatment. In April, 1902, he one day lost power in his left arm; next day his whole left side was affected; there was no loss of consciousness. He was taken to a hospital where complete left hemiplegia without impairment of tactile sensibility was observed. He was treated with potassium iodide, was discharged after four weeks as improved; the diagnosis was cerebral syphilis.

November, 1902, he retired one night dizzy and nauseated; during the night he woke up with left-sided paralysis; there was twitching of the left arm, leg and side of the face for four hours; he could not speak, was conscious, attracted attention by knocking down a screen.

He was again treated in the same hospital with sodium iodide, and hypodermics of bichloride of mercury, was discharged after 19 days, improved; diagnosis: cerebral syphilis.

After discharge he only occasionally took medicine; from time to time he had headache. In October, 1905, the patient noticed that the left leg was becoming weaker; he received glutal injections for one month. In the autumn of 1905 his work apparently was inefficient, he was discharged from the paper on which he worked. In January, 1906, he twice (on the fifteenth and twenty-second) fell abruptly without dizziness or loss of consciousness; he was able to raise himself and walk home.

He was admitted to Bellevue Hospital, January 25, complaining of having fallen in the street, of progressive weakness of the left leg, and of loss of memory. The diagnosis of cerebral syphilis was made and the patient was given potassium iodide up to 390 grains a day, and was also treated with hypodermic mercurial injections. From the beginning of his stay in B. H. he showed mental symptoms, was irrational. Soon after admission, he tried to get out of bed and take a stroll along the river. He was restless and noisy at times, sometimes mildly delirious, rambling about imaginary things. In February he had four convulsions one night. During the latter half of his stay in Bellevue he was rather irritable, and several times tried to escape. In June he was noticed to stammer badly, saw objects dimly, was generally muddled. His pupils which were noted as reacting normally on admission, were noted as irregular and slow to react in June.

On admission to M. S. H. the patient was childishly happy, affable, loquacious, amused by details, frequently laughing without much provocation. He had no grounds for his euphoria, admitted that he had "not a

d-d cent," and as to his physical condition he said, "It's a terrible plight, I don't suppose I will ever get well again"; he laughed cheerfully at the position.

He knew he was in Manhattan State Hospital on Ward's Island, gave the date correctly. He complained spontaneously of his memory being poor, and in giving his history he made several careless mistakes; these, however, he was able to correct; e. g., left school at 16, was in lawyer's office for five years, then set up in practice at *23*. He said that he had been ten years in New York, had been working twelve years for a New York newspaper; he corrected himself when the discrepancy was referred to.

He had a fair memory for the period since his first attack in 1901, although he said that the interval between the two admissions to the first hospital was 18 months (it was 6). Although his retention of a test name and number was good, he was very much confused over the incidents of the immediate past. On the day of admission he thought that he had been two nights on the island, and shortly after admission he confused M. S. H. with B. H., thought he had been there several months, said that the ward physician had vaccinated him at B. H.; and fabricated an incident of calling on the same physician at B. H.; he was confused as to the time of day; in the evening he said "I thought it was about eleven o'clock, I remember now I had dinner." He had some difficulty in giving simple facts; the name of the river Hudson, of the governor, etc., would escape him; the cause of the war with Spain was some difficulty over the tariff.

The patient admitted that he had a bad memory, but did not realize the extent of his mental impairment, nor that his mood was at all abnormal. He had no absurd ideas, admitted having had a delirious episode and hallucinations at B. H.

Physically he had the well-marked residuals of a left-sided hemiplegia—left knee-jerk more exaggerated than the right, left ankle clonus and Babinski reflex, weakness of left arm and leg, none of the face. The patient was clumsy in all movements; in walking he not only dragged the left leg, but he walked with a broad base of support, staggered from side to side. His difficulty in maintaining the erect position was increased on closing the eyes. There was tremor of the muscles of the face and of the hands; his speech was tremulous and sticking, but without transposition of syllables or distortion of the words. His writing was extremely tremulous, the words were crowded up into one corner of the paper, but were correctly written. The pupils were dilated, equal, irregular; they reacted well to light and on accommodation, also consensually. The radial arteries showed no marked thickening.

On August 14, after ten days in hospital, the patient had a series of convulsions in which the right side twitched more than the left; the convulsions left him with marked aphasic symptoms, paraphasic utterances and perseveration, and with weakness of the right arm, but without sign of Babinski on the right side. The pupils reacted very slightly to light.

The symptoms showed variable intensity for the next week, and the patient died on August 26, three weeks after admission.

The diagnosis during the life of the patient was rather doubtful, and previous to admission he had been treated as a case of brain syphilis. In the mental status one of the most important features was the presence of a striking anomaly of mood. The first patient made light of his bodily condition, talked of it as of no importance, and his optimism as to the future and as to his ability to work was evidence of impaired judgment. The second patient, on the contrary, recognized his bodily impairment—"It's a terrible plight, I don't suppose I will ever get well again"—but this intellectual recognition of his plight had no counterpart in his mood; he felt in the best of spirits, was not at all depressed, laughed cheerfully even when his condition was discussed, talked of inviting some friends to come up and take him for a drive. Wernicke has emphasized the importance, as a sign of general paralysis, of any tendency on the part of a patient to minimize physical impairment; it may, however, be well-marked in non-paralytic dementia. On the other hand this definite primary euphoria, not associated with underestimation of the physical impairment, seemed to point very strongly to a paralytic process.

The euphoria here was accompanied neither by underestimation of the physical impairment, by delusions of grandeur of any description, nor by any grandiose plans for future work.

The patient's memory of the recent past showed a partial retention of the incidents with complete confusion as to their actual sequence and relation, and with a tendency to fabricate; such a confused account, in a patient who is alert and coöperating well, is to be distinguished from the inconsistent and confused answers of a torpid patient giving poor coöperation.

The euphoria and particular memory defect, in association with the patient's alert attitude during examination, did not seem to be adequately explained by any form of cerebral syphilis, but pointed to a general paralytic process. The physical symptom-complex with the marked ataxia, difficulty in maintaining the upright position increased on closure of the eyes, marked jerky tremor of the hands and face with tremulous and sticking speech, seemed also to be explained only on the basis of general paralysis; the ataxia and the degree of the tremor were the symptoms upon which

special weight was laid. The varied nature of the neurological symptoms—diplopia in 1901, two left-sided hemiplegic attacks in 1902, the latter with inability to speak for four hours although conscious, the final convulsions with irritative symptoms on the right side followed by paralytic symptoms on the same side, and by an aphasic disorder—and the frequent headache during the course of the disease suggested brain syphilis, but did not exclude general paralysis; the negative result of treatment pointed to the latter.

On the above grounds it was felt that one was here justified in diagnosing general paralysis.

The brain showed distinct cortical atrophy over the frontal region on both sides, with well-marked ependymal granulations. The cortex showed the typical changes of general paralysis with a very pronounced perivascular infiltrate. In addition there was a cortical softening in the right second frontal convolution and an old focus of softening in the head of the caudate nucleus involving the internal capsule.

In the two cases analyzed the attitude of the patient towards his physical disorder and the nature of the memory defect have been discussed in some detail. It may be useful at this stage, for the sake of differentiation, to give a brief summary of a patient who for years was regarded as a case of general paralysis, but on closer consideration was found to be a case of organic dementia on the basis of vascular brain-disease and alcoholism.

Case 3.—Cuma G., aged 39; admitted January 11, 1901.

The patient is a Frenchman, right-handed, born 1862, of extremely alcoholic habits with a history of syphilis at 20 without medical treatment. After a trauma in 1890 he was more susceptible to alcohol than previously. For years before admission he treated his wife outrageously, did no work, drank at home. In 1899 he had an apoplectiform attack, leaving a left-sided hemiplegia; for five days he could not speak, his words gradually came back, but his speech remained seriously impaired. (The record at the French Hospital has been lost.) He was rather violent and abusive in the hospital; he was taken home after two weeks, continued to show the same behavior as before, was admitted to M. S. H. in 1901. The diagnosis of general paralysis was made on an insufficient basis; the difficulty of language and the defective articulation created the impression of a deeper dementia than really existed, and this impression was fostered by his rather fatuous chuckle.

The patient has at the present date (November, 1906), the residuals

of a left-sided hemiplegia; his speech is almost unintelligible and presents the characteristics of a pseudo-bulbar disorder. The pupils react well. His mood is one of mild discontent with detention, but during conversation he is usually good-natured, chuckles over references to his past. The special points for which I bring the case forward are the defect of insight which the patient shows and the nature of his memory defect. He appears a little surprised when the physician tells him that he is almost unintelligible, and that his gait is impaired and that he could not get work; he grins, says that he is all right and that he can again get a place as glass-polisher and earn eighteen dollars a week.

The second point is the memory defect of the alcoholic and hemiplegic Frenchman. His memory is seriously impaired; at one time he says he was married in the City Hall, New York, then again that he was married in France. He says that he was already at work during the Franco-Prussian war; as a matter of fact he was not ten at the time He gives the ages of his children incorrectely. He gives his age as 49—it is 44; the present year is 1889. When one tests his memory, he repeatedly answers "I don't know; I can't tell," seems to make no effort to remember; if one answer contradict the previous one, he is not embarrassed, makes no attempt to reconcile the two statements; he does not fumble around with numbers; the test has no interest for him, he needs repeatedly to be urged before he will give a precise number. This indifference to the test, the careless answers when much urged, the refusal to try and reconcile contradictory statements, form a different reaction from the marked fumbling with dates and unsuccessful endeavor to hold a series of correct data together, which the typical general paralytic shows.[1]

The discussion of these three cases has given rather a negative than a positive result, and suggests caution in the use of certain defect symptoms as differential points.

In the next two cases the mental defect symptoms are less marked and more difficult to use, and it is on more transient features in the mental picture and on the physical symptoms that the diagnosis rests.

The cases have not yet come to autopsy; perhaps some may hold that the diagnosis has not been established. (January, 1911. The second patient, J. W. G., remains practically in the same condition as when this paper was read and this want of progression of the symptoms makes the diagnosis still more problematical. The pa-

[1] The patient died suddenly on May 8, 1911, the neurological symptoms having shown little damage. The brain showed many foci of softening, involving the white matter and the basal nuclei; there was no evidence of general paralysis.

tient J. A. S. has shown little physical change, while an expansive trend has become rather more prominent.)

Case 4.—James A. S., 47, admitted April 25, 1905.

The patient was from his earliest years accustomed to take alcohol, and ever since his marriage in 1885 he has drunk to excess, treated his wife outrageously, gone with other women, spent most of his money on the race-track. He was a very efficient book-keeper, and notwithstanding his drunken habits he was earning $25 a week at the time of his hemiplegic attack in 1900.

When a youth of 16 or 17, he had a soft sore which healed in six weeks; he had no secondaries and a doctor said that it was not syphilis.

In 1899 he one day came home, said that he had lost himself and could not remember where he lived; there was no marked local weakness, but both he and his wife noticed that one pupil was larger than the other. The doctor diagnosed a slight stroke of paralysis. During the next year he had several transitory attacks of dizziness.

In 1900, while he was dressing, one leg became weak, he fainted. He had a left-sided hemiplegia, affecting the face and extremities. For a week or more he had great difficulty in talking, but understood what was said, knew what he wanted.

For two or three months he continued his work, but was very inefficient and was discharged; he was not drinking as it made him very nervous, nor smoking as he could not hold a cigar in his mouth. During the next two years he had occasional positions, but was usually discharged; he was unable to add up simple bills.

He continued to spend all his money on horses, none on his wife and children; he once came back from a position much excited, said that he had nearly made a million, that he received tips direct from God; he frequently made the latter assertion.

His domestic behavior became more outrageous and indecent; he occasionally left his wife; he would make silly remarks, e. g., tell his wife to dress up in gauze and waltz round. Although limping badly and staggering so that he seemed about to fall, he maintained that he was all right.

In 1903 he gradually became completely deaf in both ears. Owing to his dilapidated behavior he was finally certified as insane.

During the eighteen months spent in the hospital the patient has shown no change. According to his wife he was always an accomplished liar, and in hospital the patient tells a plausible story, admitting his alcoholic and gambling propensities, but denying that he ever talked of receiving tips from God, that he talked of making a million or that he ever behaved outrageously. He gives a good chronological account of his life with exact dates and no discrepancies. He converses rationally (the physician has to write his questions), takes an interest in the daily papers, remembers incidents well, has a fair grasp of current events, and ordinary information; he adds promptly, calculates the equivalent in British coinage of $385 with only a slight mistake.

Physically, he is a well-built man, 48 years of age, with exaggeration of all the deep reflexes and residuals of the left-sided hemiplegic attack in 1901—weakness of the left side, left-sided ankle clonus, double Babinski, no dulling of sensibility on the left side. In addition to the mowing gait of the hemiplegic he is markedly ataxic; closing the eyes increases his unsteadiness in the upright position. The pupils are unequal, irregular; the right is Argyll Robertson, the left reacts rather sluggishly to light. The speech is loud and slurring, but not sticking nor tremulous. His writing is on the whole good, but there is occasional untidiness, omission and repetition of a word or letter or mistake with a syllable, e. g., Epis*p*ocal, charter*n*ig, resig*e*nd, practi*l*ly. He cannot name the test solutions, but smells equally well on the two sides. He occasionally hears a very loud command; he hears several noises on the ward. There is a well-marked lymphocytosis of the cerebrospinal fluid.

In this case, as in the first, the development of the disease was masked by alcoholism, and the patient still has the attitude of a plausible alcoholic liar. This somewhat complicates the estimation of his insight. He wishes to go out, says that he wishes to work for his wife, and that old employers may help him to get some position. While he recognizes his deafness and the presence of weakness on the left side, he does not admit any mental impairment nor have an adequate idea of his physical condition; his wife had been struck by his refusal to admit that there was anything the matter with him.

The patient's marked alcoholism accounts in part for his abnormal behavior before admission, but not for several factors—the absolute loss of the sense of decency (the patient would defecate on a chair instead of going to the toilet; he would, when apparently sober, sexually assault his wife in presence of the children), the childish behavior (e. g., he would throw into the box a letter with a farewell message to his wife, and would then run away), his receiving tips from God and talk of making millions (at this time he was not drinking hard). This behavior had a peculiar stamp of dilapidation; it is met with in general paralysis, and I do not know if cases of brain syphilis may present it.

Mentally, then, the important differential points in this case are the special form of dilapidated conduct, the inadequate realization of his condition, the psychotic symptoms, and the mistakes in writing. The condition of the pupils may be met with in brain syphilis; as to the value of the ataxia with definite sign of Romberg as a differential point between general paralysis and the various forms of brain syphilis, I have no data.

Case 5.—Joseph W. G., 38, admitted July 30, 1901.

The patient was born in 1864 in New York city, developed normally and worked at a variety of occupations; he is not known to have taken alcohol to excess. In 1890 he had a sore on his penis, and took internal

medicine for one month; in 1894 he began to suffer from pains in the back and chest, accompanied by nausea and vomiting; for the next six years he continued to have episodes of lancinating pains at intervals of two and three weeks; in 1900 he was diagnosed incipient locomotor ataxia; the pupils were Argyll Robertson, the knee jerks diminished.

For a few months before commitment in 1901, he had grandiose plans for working a patent, he talked a great deal about money, and, when committed, he was talkative, elated, and expansive; he said that he was worth $90,000, the patent was worth $35,000,000.

After a few months, this trend simmered down and the patient became slightly depressed; one year after admission, he had much improved and possessed considerable insight into his past condition.

He had later a depressed period, then in 1904 was megalomanic.

*Neurological Incidents.*—The patient had already in 1898 had a transitory weakness of his left side with paraesthesia, of half an hour's duration; in 1903, he had an attack of unconsciousness followed by confusion; in 1904 he had a sudden apoplectiform attack, leaving a permanent left-sided hemiplegia with marked impairment of sensibility on the left side, and with left-sided hemianopia.

*Present Condition,* November, 1906.—The patient has, for at least a year, shown practically the same condition; he is quite clear, knows all the hospital gossip, takes an interest in the papers, wishes to go home; he admits that he had temporary insanity on admission, sometimes he says that it was the commitment which made him insane; he says that he was funny, had strange ideas in 1901; he attributes his megalomanic attack in 1904 to being tormented by other patients. He misinterprets various paraesthesias as due to electricity and magnetism, and frequently shouts out loudly at night owing to this torment; when questioned on the subject, he is evasive, at first merely talks of an uncomfortable mattress, then admits that the feelings must be due to electricity, etc.; there is little attempt to elaborate the subject. He says that he is constipated; when urged, he makes hypochondriacal statements as to his abdomen, his stomach is all cut out, he is ruined, he is only a shell, the attendants during his crazy spell in 1904 cut his stomach out with poison.

The patient has an excellent memory, both for the recent and the remote past, he has a detailed grasp of his various transfers in the hospital and of the dates of minor events; there is no discrepancy in his dates.

*Physically,* the patient shows the residuals of a left-sided hemiplegia; there is little weakness, but there is Babinski reflex on the left side, hemiplegic gait with the arm in the wing position, the sensibility much more impaired on the left than on the right side; left-sided hemianopia; there is marked ataxia of left arm and leg; the difficulty in maintaining the upright position is not increased on closing the eyes; the knee jerks, which were diminished in 1901, are now definitely exaggerated; the pupils are equal, irregular, Argyll Robertson. Writing tremulous with distorted words. Speech tremulous.

The patient realizes his physical impairment, he attributes it to his

treatment in the hospital, misinterprets his abnormal sensations; he is anxious to go home and help his mother, says that he can get a job as night-watchman, in some places the watchman needs only to sit behind the door all night, he could make money as an entertainer as he is full of jokes, he could write a book of experiences, and would easily find a publisher.

This self-confidence is based rather on a want of judgment of the ordinary relations of life than on a failure to recognize his own general condition; it is the same symptom, though in less degree, as his original grandiose plans to develop a patent.

He admits that he is physically weak and that he is even slightly touched in the head; fresh air and city doctors will soon cure him.

The patient's want of judgment is seen in another point; he frequently sits and loudly talks of the injustice of his detention; when interviewed, he frequently repeats his grievances, but he does so more apparently to give vent to his feelings than with any hope of changing the situation.

He never argues the point with the physician, never asks him to take definite steps for his discharge nor asks him for the grounds of his detention; while repeating his claims, he acquiesces in the situation, sometimes even laughs at it and bears no ill-will for his detention.

This acquiescence in the situation in certain cases of dementia paralytica contrasts strikingly with the retained intellectual grasp of relations and with the actual demands for discharge, which may be set aside by the physician without provoking any remonstrance from the patient. Besides these defect symptoms the patient has presented additional symptoms, megalomanic periods, persistent hypochondriacal statements, and false interpretations of morbid sensations. The importance of the former two elements in the diagnosis of general paralysis is generally emphasized: one must remember, however, that in syphilitic meningoencephalitis a megalomanic trend may be present, and a hypochondriacal element is frequently present in arteriosclerotic dementia.[2]

Case 6.—Mrs. Annie F., 43, admitted March 18, 1905.

The patient is a Russian woman of low social status, who was an efficient housewife, and enjoyed moderate health until 1902 or 1903; no history of syphilitic infection was obtained from the informants, but the patient had numerous suspicious scars on her legs; she had several miscarriages and her husband has suspicious nervous symptoms. About three years before admission, she began to eat ravenously and to sleep constantly, she neglected her housework, was very apathetic, complained of frontal headache and of seeing double. On one occasion, about two

[2] The patient died on May 28, 1911 from a sub-dural hæmorrhage caused by a fall during a convulsive seizure. At the autopsy the brain presented a diffuse atrophy of the right hemisphere, but no definite evidence of general paralysis: microscopical examination disclosed the histopathological changes of general paralysis.

years before admission, she dæfecated in bed, then threw the fæces with her hand on the wall; when asked what was the matter, she said "nothing"; during the last two years, she spent the whole time sitting in her chair, took no interest in what went on, admitted that she was weak, but said that there was nothing wrong with her; she answered most questions with "I don't know," she occasionally soiled the bed. There was no history of elation nor depression; a vague statement that she had heard voices was not corroborated.

*On admission* the patient was very placid, had a never-failing mechanical smile; she answered questions pleasantly, but showed little spontaneity; her mood was one of complacent indifference; she accepted the environment without criticism; there was no elation, no definite depression, but she felt that she was an invalid, emphasized her ailments with some hypochondriacal zest; "I am very weak—I am nearly blind—I can't walk." She gave a strong handgrip; her gait was shuffling, but not very much impaired; she could count correctly fingers held up at ten feet; at the same time she said that she saw double.

The patient showed a marked memory defect, which consisted not so much in loss of memory of the incidents as in inability to space them out correctly in time; successive answers to the same questions were widely different, e. g., she said that she sailed for America at 15, 19 and 29; with regard to other events, she gave equally inconsistent dates. She did not spontaneously see even glaring discrepancies; she said that she was divorced at 23, came to America at 25, came to America in the year of her divorce. She was an extremely ignorant woman, but her calculation was better than one would have expected.

The patient realized her changed mental and physical condition; "I am not clear—I have not the spirit of work—I should like to sleep always for the last two years—my head is so slow—my head is weak"; she exaggerated rather than minimized her physical ailments.

*Physically,* she was a well-nourished Jewess with fair muscular force and no sensory disorder; the knee-jerks were both exaggerated; the gait was shuffling, not ataxic; sign of Romberg was noted on the first examination, but not on subsequent examinations; there was tremor and jerking of individual fingers; the speech showed decided sticking on l and r with tremor of the facial muscles; the pupils were equal, reacted well; weakness of left external rectus and slight nystagmus in external fixation. She complained of a variety of subjective disorders during the previous two years—severe frontal headaches, noises of whistling and ringing in the ears, her head felt funny, she was like drunk, was dizzy.

During the nineteen months of hospital residence, the patient has shown little variation; she does whatever work she is asked to do, keeps track of the time and of the major incidents in the ward; during an interview she asks for discharge, but never pushes the question nor spontaneously addresses the physician with this in view.

In this case, as in the preceding cases, there was a dementia

characterized by an inadequate attitude towards detention and by a striking inability to see glaring discrepancies in dates; additional features were the episode at the onset and the mild hypochondriasis.

I do not know that cases of brain syphilis ever present such a form of dementia.

*Tabes and General Paralysis.*—Where a psychosis is found to be associated with tabes one is tempted to immediately conclude that the case is one of general paralysis. In several cases reported in the literature, where this was not done and a non-paralytic dementia with tabes had been diagnosed, the anatomical examination demonstrated the histopathological changes of general paralysis. Many cases have been published of non-paralytic psychoses with tabes; there is on the one side the case where a patient with a psychosis, e. g., a manic-depressive, gets syphilis and later develops tabes, and, on the other side, the case where on the basis of an established tabes a psychosis, usually a paranoic condition or a depression, arises.

Alzheimer records two cases where the dementia was demonstrated to be not due to a general paralytic process.

In the following case, for a long period it was felt that there were insufficient grounds for considering the disorder general paralysis; the nature of the dementia which later became established cleared up the diagnosis.

Case 7.—Mary B., 46, admitted February 22, 1905, discharged December 24, 1905; readmitted May 5, 1906.

The patient is an Irish woman who has lived a rather irregular sexual life, and whose husband claims that he received syphilis from her in 1899; there is no history of alcoholism. On the death of her youngest child in October, 1904, she was rather depressed, but seemed to regain her equilibrium. In February, 1905, she began to complain of being sick and of pains in her legs; she was sleepless, refused food and medicine. Two weeks later she made peculiar treading movements with her legs in bed, said she could not help it.

For several days before admission she said "I think I'm getting crazy." She told the ambulance surgeon that worry over her baby had driven her crazy. In Bellevue Hospital she uttered ideas of fear with smiling unconcern; she said "I see all kinds of funny things when I close my eyes, I'm so light-headed all the time, I hear kind of funny noises in my ears."

On admission the patient was quiet, seemed to take no interest in her

surroundings, showed no spontaneity; she answered questions in monosyllables, replied "I don't know" to almost every question, even to questions to which she had previously given a correct answer. Her mood, when unmolested, was one of complete indifference, but when examined vigorously her unconcern was replaced by a look of dazed perplexity and some alarm and agitation. Owing to her monotonous answer of "I don't know," or "I can't tell" to almost every question, her memory, grasp of general information, etc., could not be tested.

*Physical Status.*—Absence of knee- and Achilles-jerks, also of corneal and pharyngeal reflexes; very slight reaction of pupils to light, fair on accommodation; unsteadiness of gait and upright position; no reaction to pin-pricks except over the soles of the feet. The mouth was foul, the tongue dry and hard.

For two or three weeks the patient continued to give the same negative answers; she made no sign of recognizing her children. She said that she was blind, but at the same time she recognized figures and letters. One day she said that she had no feeling, no life—"you have no idea how terrible I feel, I wish I was dead rather than in this way." On one occasion she recognized her children at first, but during the same interview said that she did not know them; when asked to explain this she said, "I could not see them—I could not see them plain."

In April she improved, took some interest in home affairs, was more cleanly; for two weeks she frequently walked with her legs crossed, even when going up-stairs; no reason was given for this. She winced at pin-pricks, but denied that she felt pain. She began to answer questions about the past, but was absolutely indifferent to absurd contradictions in her time relations; she remembered in remarkable detail the events associated with her admission, having registered correctly even the names of the patients who came with her from Bellevue Hospital to M. S. H.; for a while she was depressed over her condition, which she described as incurable insanity. Although she had a fair grasp of the environment, she thought the city was "empty houses," the people on the passing steamboats were "make-believe."

Her memory improved so that she was able to give a fairly accurate account of her life. In December she was well enough to be discharged. Her plans for the future were quite reasonable; she had good insight into her mental disorder, remembered the various peculiar ideas she had had. Physically there was noted absence of knee-jerks, Argyll Robertson pupils, slight difficulty in speaking the test phrases; no tremor, no ataxia, no sign of Romberg.

In view of the close connection of tabes and general paralysis the diagnosis of general paralysis seemed the most probable one, but in view of the peculiar nature of the mental symptoms the diagnosis was not considered as established, nor the possibility of a non-paralytic tabetic psychosis excluded. The mental picture had been that of a depression, with a consciousness of mental inefficiency, which later passed into a peculiar condition of apathy with negative replies; when she improved

so far as to give her history, she ignored the hopeless discrepancies in her statements; on further improvement her discrepancies seemed almost sufficiently accounted for by her general ignorance. The possibility of an hysterical element was suggested by the apparently blank state of mind, the abrupt alternation between recognition and non-recognition of visitors, the denial of any memory of a visit with no indication of an actual defect of retention, a certain suggestibility, and the peculiar gait which seemed to arise from a casual attitude; the pharyngeal reflex was absent, the corneal much diminished, there was almost general analgesia.

The diagnosis was therefore not considered to be established.

The patient was discharged on Christmas Eve, 1905, and appeared to be in good mental health, but after about one month she began to show marked lapses in conduct; she would go about the house practically naked, she wandered away from home for days at a time; she was, therefore, readmitted May 5, 1906.

On re-admission the patient recognized all her old acquaintances, said that she was glad to be back, talked of her sickness with cheerful indifference, gossiped pleasantly about the past. She described in a matter-of-fact way an episode when she had rambled away from home and spent two days in the park: "I don't know why I stayed—I guess I was too lazy to walk home." She compared herself jocularly to an actor out of work. She admitted that this was crazy behavior, saying "sure, I was off my trolley again," but again said that she was not crazy.

She had a good memory for the incidents of the past, but showed absolute inability to grasp glaring discrepancies in her dates; she said "this is 1896," and that she had her last baby July 4, 1896.

Her conduct in the ward was rather dilapidated; she was very uncleanly, at night pulled at the other patients' clothes as she wanted to go home; she pinned bed linen under her own clothes. The garrulous talk of the patient was of a drifting flighty character, e. g., "Do you know my sister Nellie is dead, God is slow but he's sure, we all have to go some day, how is it the Chinese get something to eat in the coffin, they wake up and have something to eat (laughs), the Jews put in a quarter, she was buried in Evergreen with her husband," etc.

She commented on passing boats and on remarks of other patients.

Her writing was much worse than during the previous admission, the words being distorted into a series of meaningless characters.

After about a month the patient had given up her pranks, was cleanly and a useful worker, and since that time she has continued to maintain the same level of a mild dementia. She knows all the hospital gossip, enjoys life in the ward but talks of returning home again; her future plans are quite reasonable. She still shows absurd discrepancies in her dates, e. g., married in 1885, born in 1896. She admits that she was crazy on her first admission, but not on the second; she was only brought here because she went to the park.

The following are the important features in her dementia—the

contentment with hospital life as satisfying most of her aspirations, the uncritical attitude towards the wandering episodes and past behavior, the absurdly contradictory dates, the extreme distortion of written words; such a dementia presents the features which in the preceding cases were emphasized as of differential importance, and in conjunction with tabes warrants the diagnosis of dementia paralytica.

From the above observations it would seem that for clinical differentiation a study of the dementia itself is important. The dementia, however, was not the only element present in the cases; other features were also present, either behavior with a peculiar stamp of dilapidation, or psychotic symptoms, which form the "crazy" element in the picture.

The limits of this communication do not permit a discussion of these latter symptoms, but it is sometimes on these latter symptoms that our diagnosis frequently rests; the diagnosis may be established before the presence of any dementia can be demonstrated; no defect may be observed save the absence of the patient's critical faculty in face of his own grandiose plans.

This was the case in the following observation.

Case 8.—The patient (Emil K.), admitted January 13, 1906, age 35, an alcoholic bartender, became restless and irritable shortly before admission, was unable to do his work, formed great plans for making money, was sent to the hospital. When admitted to the hospital he was restless, excited, talkative, harped on his grandiose schemes: he intended to marry a beautiful girl, would call on the Emperor, etc. He showed absolutely no memory defect, was clear in his orientation. His restlessness quickly disappeared, the expansive trend simmered down, and two months after admission there was no trace of the psychosis, except a slight tendency to minimize the absurdity of his crazy ideas on admission.

He was discharged four months after admission apparently in excellent mental health, took up a position and earned fair wages.

His physical status presented the same neurological complex during his whole stay in the hospital—exaggeration of deep reflexes, unequal pupils reacting sluggishly to light, tremor of fingers and tongue, marked lymphocytosis of the cerebrospinal fluid; no defect in speech, writing nor gait.

Here, then, was a fairly pure megalomanic episode without any of the defect symptoms described above. The diagnosis, however, did not seem open to doubt.

In such a case the presence of a well marked lymphocytosis is a source of comfort to the clinical observer.

The last case which I wish to report is that of a patient observed by Dr. Meyer, where the possibility of brain tumor was seriously considered, but which turned out to be an atypical general paralysis.

CASE 9.—The patient was born in 1857, at the age of 23 had some venereal infection, when 42 complained of unsteadiness and weakness of the left hand; he became fatigued, unable to do his work; his gait became unsteady, with weakness of the left leg. After three months' vacation he became slightly expansive, irritable, and showed a childish behavior.

January 17, 1900, twitching of the left arm, anesthesia of the left arm, reduction of muscular sense and attention to motility on the left side, exaggerated tendon reflexes on the left side. The anaesthesia spread later to the trunk and the left leg; Babinski reflex developed on the left side. In October the patient had an attack of left-sided twitching; after this, left-sided hemianopia was noted. The left pupil was larger and more sluggish than the right. The left side became more rigid.

As to the mental symptoms the patient had been euphoric and talkative at first, with good orientation, fair memory, but defective calculation. In summer he became dull, careless and untidy. In November he fabricated; he later became duller and less responsive. He died May, 1901.

As to the diagnosis, the optic discs were normal, there was no history of headache, vomiting nor dizziness, but both the irritative and the paralytic symptoms with their steady progression suggested the possibility of a tumor. The mental symptoms suggested strongly general paralysis, but were not inconsistent with brain tumor.

On post-mortem examination it was found to be a case of atypical general paralysis, the process being of exceptional severity over the posterior half of the right hemisphere. In some parts of this region there was complete disappearance of the nerve cells; the vessel changes were well marked, the perivascular infiltrate moderate. On the right side the pulvinar was much affected. In addition to the severe affection of the right parietal and occipital lobes there was present a corresponding atrophy of the left cerebellar hemisphere.

# CLINICAL VARIETIES OF PERIODIC DRINKING[1]

By Pearce Bailey, M.D.,

NEW YORK

Singled out and separate from habitual drinking is a type of alcoholism characterized by its periodicity. There are recurring attacks of intemperance and debauch, lasting from a few days to several weeks. Between the attacks the subjects are either temperate or abstemious, or have a distinct distaste for liquor, and, for the first few years at least, bear none of the physical or mental stigmata of alcoholism. The periodic character of this variety of inebriety has long caused it to be compared with another paroxysmal disease, epilepsy. And since Gaupp, in a carefully prepared monograph on dipsomania, published in 1901, drew the lines closely together from cases of his own and from literature, there has been a gradually growing conviction that dipsomania is one of the larval forms of epilepsy. It cannot be denied that there are striking points of similarity between typical cases of dipsomania and epilepsy. Many dipsomaniacs have had convulsions and in nearly all of them may be found the same neuropathic antecedents as are met with in the histories of epileptics. Again, the action of alcohol upon a diseased or intolerant brain recalls in many ways certain symptoms of epilepsy. Intolerance to alcohol, to the results of which the Germans have given the name pathological drunkenness, is essentially epileptic in character. In it, after the ingestion of very small quantities of spirits or even beer, the individual becomes immediately flushed and excited, violent and often dangerous to others. A similar intolerance is common in epileptics, and is particularly liable to induce states of automatism in which the patient is no longer responsible. It is also frequent in other cortical diseases or degenerations, of which it may be the earliest diagnostic sign. Notable among the brain

[1] Read at a meeting of the New York Psychiatrical Society on May 5, 1909. 

conditions in which intolerance is encountered are general paresis, degeneration of the cerebral arteries, and the state of depressed cortical function which often follows traumatic injuries of the head.

In addition to these general similarities an attack of dipsomania has certain psychic characteristics in common with those of grand mal. In both the patient becomes, for a short time before the attack, restless, anxious, dissatisfied; he reproaches himself for misconduct which had long lain forgotten; and all this without reasonable cause. This premonitory depression is constant in true dipsomania and is becoming more and more recognized as a cardinal symptom of epilepsy. In addition, retrograde amnesia, as well as amnesia for parts of the attack itself, is common in both conditions.

I have notes of a case of a young man, by nature temperate and self-controlled, in whom the attacks recurred every two or three months, sometimes less frequently. The antecedent depression was marked, and readily recognized by the family. The seizures were characterized by wild intemperance, debauch, and violence. There were distinct losses of memory for parts of the attack itself, and often also for several hours preceding the first drink. The apparently hopeless outlook in this case, which I have watched for five years; its explosive character without any psychic cause which can be ascertained, and the bad family history, seem to mark it definitely as dipsomania of epileptic nature, dependent on the same cortical irritation as causes epilepsy. Certain cases cited by Gaupp also seem equally incontestable. But such cases are rarities.

Perhaps no one sees so many varieties of alcoholism in their formation period as a neurologist practising in a large city. And of the various cases of periodic drinking I have seen, the one mentioned is the only one in which epilepsy seemed the only explanation. In the others, various causal factors pressed forward for recognition, equally or more deserving of attention than epilepsy. So I now believe that many of the so-called epileptic dipsomanias can be better explained on some other hypothesis; that what seem at first sight as epileptic explosions can frequently be reduced to certain phases of mental disease, the clinical characteristics of

which soon become blurred by alcohol, or to the influence of some recurring psychic motive.

The importance of such a distinction is real, both for prognosis and treatment. True dipsomania has the same prognosis and treatment as epilepsy. Periodic drinking from other causes offers more hope for amelioration in that the cause, when found, is more get-at-able; and the case as a whole can be better understood and more rationally handled, if its causal factors are revealed.

In the listed types of mental disease, alcohol plays a varying rôle. It is not conspicuous in dementia præcox or in melancholia. In fact, the generally received belief that mental depression causes periodic drinking seems true only insofar as it applies to depression within the limits of sanity. Under the stress of business reverses, family troubles, failing health, many become tipplers. Some become periodic drinkers. The most familiar type of this is the emotional, high strung man who gets wildly drunk, often for several days, after quarrels with his wife. But the recurring attacks of depression in which the depression is out of sane proportion to the causes alleged to have induced it—that is, the recurring depressive cycles of a manic-depressive insanity, seem rarely if ever to incite to inebriety.

In general paresis sudden attacks of inebriety are extremely common, as is to be expected from the epileptic associations of this disease. It is only when these attacks precede the physical signs or the mental deterioration that diagnosis is difficult.

In the manic phases of a manic-depressive psychosis, periodic drinking occurs in a way to obscure the clinical picture. In these cases the mental disease is accountable for the inebriety; but the effects of alcohol so far change the ordinary clinical behavior that it is usually only after two or three attacks that the true nature of inebriety is discovered. The two following cases illustrate this:

One is that of a young man who has been committed to Bloomingdale thirteen times in the past ten years. I appeared in one commitment, but for details of the history I am indebted to Dr. S. B. Lyon. The original diagnosis was dipsomania, and the patient's wife still believes alcohol to be at the root of all the trouble. In the beginning of the attacks while drinking, he is confused, delusional, and disordered, with more or less excitement. During the attacks, he is abusive, denounces his commit-

ment as unjust, and threatens court proceedings. Before some of the commitments, he has been violent and made assaults. On one occasion he improved sufficiently to be removed by his mother. Two days later, he was drunk again with return of mental symptoms—he was dirty, disheveled, excited, garrulous, talked rapidly, profanely, and disconnectedly—a condition lasting several months.

Another case of this character is the following:

A man, 37 years of age when he first came under observation, had proved himself of more than ordinary business capacity. He had built up a successful business and had made shrewd investments. At college he had been regarded as intemperate. He used to go off on sprees, though not a regular drinker, and acted queerly. He was married in 1904, the bride being aware of his reputation. Shortly after his marriage, he refused for a time to speak to his wife, felt that he had lost all his money, felt that there was no hope for him, and threatened suicide. During this time he did not drink at all. In August, 1905, he began to drink and was intoxicated most of the time. At the same time, he became expansive. He bought a great many things that he had no use for, though he almost always got good bargains. A piece of real estate that he bought at this time was sold at double the price he paid for it. He would play the piano all night and would go out in his night clothes at four o'clock in the morning to feed the dogs and chickens. He kept the whole house awake with his orgies, drinking constantly; he would lock himself in the wine closet for hours at a time. Though very drunk, he appreciated fully the object of a visit from two examiners in lunacy and swore he would not be locked up. Placed in confinement, the evidences of alcoholism rapidly left him and he remained in a condition of mild reasoning mania. He reasoned with such skill that no sheriff's jury would have held him, and as he insisted on liberty or an *inquirendo,* he was discharged. He immediately became much depressed, bore no ill will toward those active in his commitment and did not drink at all. Later he resumed drinking wine at dinner without apparent ill effect and without intemperance. For three years he was well, temperate, successfully engaged in business. There was one period of excitement and intemperance which lasted several weeks, but for which no special medical attention was necessary. In the autumn of 1908 he

began to drink again. He would insist on making several cocktails for everyone who called; he wrapped a napkin about his waist, served all drinks himself and said he was the butler. And, as before, would lock himself in the wine closet for hours. He developed persecutory ideas. Thought that detectives were after him, that the people on the street were making remarks about him, that people in neighboring houses were mocking him. He was placed, without commitment, in a private sanatorium. He thought he was committed and would not cross the threshold of his room in the fear that the police would get him. He had developed some important signs of alcoholism. The knee jerks were absent. And another sign, the occurrence of which as an alcoholic symptom is overlooked in most text-books, was that the pupils did not respond to light. After a few weeks' abstinence, both the knee jerks and the light reflex became normal. But he still was insane and was committed.

He was filthy in his habits and would spend his days making messes of food and odd bits of rubbish, which he called inventions. He was confined altogether for about two months, at the end of which time he was well enough to be discharged. He again became depressed, all delusions left him, and he had no desire to drink.

Both of the above cases seem to have classed themselves pretty definitely as manic phases of manic-depressive insanity. By some peculiarity of personal disposition or environment the expansive periods announced themselves by sudden outbursts of inebriety; and the resulting alcoholism marred the clearness of the mental picture. But the diagnosis was long in doubt, and by the lay mind both patients still are believed to be periodic drinkers.

Outside the sphere of well defined psychoses are many mental states touching the abnormal and characterized by instability, by impulsiveness, by excessive psychomotor reactions. It seems reasonable to think that further examination in this field may throw much light on periodic drinking.

Many of the psychic causes and the psychic effects of alcohol are interchangeable. In studying the mental states which lead to drinking, we may find one that seems important, and mark it down as an essential cause. Then, later, when observing the effects on character of alcoholism, we come again, with startling frequency,

upon the same feature which caught our eye when studying causes. It may have become, perhaps, discolored, and present lines more sinister, but is none the less unmistakably the same as we knew it as a cause. Take, for example, fear. Fear as a cause of drinking has become a proverb, and so requires neither exploitation nor comment. As a symptom, fear is disseminated through the whole clinical fabric, from the timidity of the besotted vagabond to the wild terror of the victim of alcoholic hallucinations. Thus fear is both a cause and an effect. So it is with many other of the factors busy in the genesis of this world disease. We see them as causes and, shortly afterwards, they are looking at us branded as effects. Like sheep at pasture, they jump their boundaries.

Sexual desires, wrong moral attitudes, idleness, jealousy, all appear indifferently in the category of causes or effects. And in studying the springs of inebriety, we may do worse than begin with the effects. Two of these latter—sexual excitement and jealousy—deserve especial scrutiny. The relationship which exists between the sexual appetite and the stimulant which best arouses it needs only to be mentioned to be recognized. Indiscriminate license, sexual perversion, sexual crimes, all are the results of intoxication, as readily appears in every treatise on psychiatry and legal medicine. But that certain forms of alcoholism owe their existence to sexual desire is not so well established. Normal intercourse has nothing to do with drinking; and the alcoholic hilarity which enhances the popularity of the brothel stimulates desire rather than results from it. But in periodic drinking, the generative feature stands out more closely. Procreative tendencies are themselves more or less periodic in their appearances; and their impulsive character is revealed by such degenerates as exhibitionists and curl cutters or by such imperative ideas as are found in the psychoses which result from sexual traumata in childhood. I have yet to meet a periodic drinker who was not an erotomaniac as well. One patient whom I have observed for fifteen years and who has been incarcerated in almost every institution within 100 miles of New York has never gone on a spree from which harlots were excluded. With him, contemporaneously with the idea of the initial drink, came the idea of sexual gratification. And when his family went to look him up, they would always find him in a

house of prostitution, never in a bar-room. It is true that in many dipsomaniacs, the erotic ideas do not make their appearance until the stimulant has aroused desire. But in the case just described they appeared as soon as, if not sooner than, the impulse to drink; and there would be no difficulty in mustering other cases of this class. So it seems worth while to give attention to the hypothesis that some cases of dipsomania arise in the sexual centers rather than in the motor cortex—and that they are a part of a general neuropathic state and are psychogenic in origin, having no direct relationship to epilepsy. Cases in this class distinguish themselves from epileptic dipsomania in that aggressive physical violence is not conspicuous during the attacks; that the attacks can persist over many years without pronounced mental deterioration; and that, as time goes on, the attacks may become less severe and less frequent.

Another variety of periodic drinking is sometimes met with in personalities mildly paranoid, usually of the jealous type. Jealousy is among the most frequent of the psychic symptoms of chronic alcoholism, and consequently when one encounters a case of alcoholism with jealous delusions one is apt to infer that the delusions are toxic products. But this is not always correct. In a number of cases that have come under my notice subsequent events have shown that the periodic drinking was a fortuitous circumstance. The paranoid state was amplified by it, but existed independently of it—and in one case in particular was only moderately intensified by the alcohol.

This case was that of a lady of highly neurotic temperament and a jealous disposition. After having borne her husband several children she became infected with syphilis. The infection is believed to have occurred at a gynecological operation. The husband never had syphilis. But the occurrence of the misfortune intensified the wife's jealous state of mind. She wished to know everywhere her husband went; would upbraid him for his alleged attentions to other women; and frequently insulted different female members of her own family on the ground that her husband was unduly attentive to them. Her attitude toward her husband was fluctuating; at one time solicitous and affectionate, at another she would work herself up into a jealous rage, upbraiding him and accusing him of all manner of improper

acts of which he was guiltless. On these occasions, she would take to drink, which intensified her fury. Once she developed a typical jealous mania. She went to the police and set detectives on her husband's trail; interviewed the newspapers; threatened divorce; put a truly insane interpretation on the most trivial circumstances; said she would ruin her whole family if necessary, but that her husband should be exposed. She was drinking heavily at this time and those who saw her—police, detectives, and members of her own and her husband's family—all thought her action the sole result of drink.

Placed under treatment and being brought to realize that none of her contentions would be believed by anyone if she drank, she stopped drinking, and though she stopped abruptly, showed none of the physical signs of alcoholism. But the mental symptoms kept on in full activity, and for months afterward, although she was totally abstemious, the psychosis continued in full flower.

Another promising field for future investigation as to the genesis of periodic drinking should be among the class of emotional personalities broadly embraced by the terms hysteria and psychasthenia.

Throughout the clinical range of alcoholism, both of the single intoxication and of the chronic poisoning, there is disturbance in the emotional sphere. Individual feelings such as anger, grief, joy, fear, attain undue prominence, and react to stimuli too easily. This fact explains why cures, whether they be religious, "scientific," or commercial, which appeal to the emotions, are the ones which have the greatest success with the drunkard.

And as we find these psychic features as results, it would not be surprising if they also figured as causes. We know now that many obsessions, tics, morbid fears, and even certain paranoid states, had their starting points in some painful emotional experience. I believe that periodic drinking, allied in many ways to these psychasthenic or hysterical complexes, will soon be shown, in many instances, to have had a similar starting point.

I regret that no case in my records has been analyzed from this point of view, although in one case, seen many years ago, there seemed an intimate connection between the paroxysms of drinking and certain feelings of inadequacy, which latter resulted from unfortunate surroundings in childhood. The newer meth-

ods of psychoanalysis would perhaps have shown a still closer relationship.

In closing, I would urge a careful psychological analysis of all cases of periodic drinking. It is only by such means that the proper curative measures can be unearthed. Chronic alcoholism has some right and title to be considered a disease. But dipsomania, in most cases at least, is not so much a disease as it is disorder of personality. And the treatment must be shaped to that end. Before this is possible, the defects in the personality must be laid bare. The treatment, therefore, is individualistic, varying in every patient in accordance with the result of the analysis of him as an individual.

# A STUDY OF SOME CASES OF DELIRIUM PRODUCED BY DRUGS[1]

By Dr. August Hoch,

PSYCHIATRIC INSTITUTE, N. Y. STATE HOSPITALS

Cases of the nature of those here recorded are probably not very rare. Nevertheless, during my ten years' service at the M'Lean Hospital, Waverley, Mass., I have had occasion to observe only eight, four of which are here presented. But it seemed to me of some value to establish clearly the delirious nature of these conditions, to analyse them carefully, and to compare them with the deliria about which we are best informed, those produced by alcohol. The excellent monograph by Bonhoeffer,[2] a model of clinical analysis, has greatly advanced our knowledge of delirium tremens and of deliria in general. The desire was very natural, therefore, to study deliria with a different etiology in a similar manner. That the writer feels a great obligation to Bonhoeffer, whose work in part guided his studies and his conclusions, he desires to express at the outset.

The drugs to which these deliria were attributed are chiefly bromides, hyoscine, various true hypnotics, and morphine, and it is a notable fact that it seems to be of very little consequence which drug is used; indeed, I have seen one case in which acetanilid seemed to have been the only, or at any rate, the most important drug. After all, as is assumed in the case of alcohol, the action of the poison introduced is probably only the indirect cause; nor does it seem to be the only one, for insufficient food, protracted loss of sleep, digestive disorders, and general exhaustion, seem to act as contributory causes. We may infer this from the fact that such factors are often present, and that we find occasionally conditions resembling delirious reactions in manic states, for example, after just such causes have

[1] Read at the New York Psychiatrical Society, October 4, 1905.

[2] Bonhoeffer, Die Geistesstörungen der Gewohnheitstrinker, Gustav Fischer, Jena, 1901.

been at work. Unfortunately it has usually been impossible to determine the exact amounts of the drugs taken, and in one case the doses admitted seemed too small to account for the profound reactions. Nevertheless, the experience with all such cases cannot leave any doubt regarding the importance of drugs as an etiological factor in them.

Case I.—Mrs. H., aged 51. In the hospital from March 5 to March 28, 1903. The patient had one sister who had the opium habit. Any other neuropathic traits in the family were denied.

The patient had never been insane, but since the age of 30 had complained of very severe headaches which occurred at menstruation, and for years had been in the habit of taking morphine for them to the extent of ⅛ to ½ grain a day. She is said to have been perfectly well in the intervals. For three months before admission the patient had not menstruated, after the flow had been scanty for about a year. Two months before admission the headaches again came on, and now became continuous; she took morphine, rising rapidly to a grain a day, but, it is said, no farther. This was continued until admission, while in the meantime bromides were added. These, it was claimed, were not in large doses. The patient had become irritable, and two weeks before admission she began to get restless, somewhat apprehensive, and for five or six days before admission she is said to have been confused and at times dull. For a week she had not slept and had scarcely taken any food.

On admission the patient appeared restless, evidently heard voices, but she showed no fear. She was disoriented and used wrong words. At the morning visit on the following day she was found with a rather pasty complexion, a heavily coated tongue, a temp. of 99.2°. Her breath was foul. There was no eruption on the body. There was no evidence of any palsies; the movements of the arms were not ataxic, but the gait was rather staggering. There was a general coarse tremor in the hands. The reflexes were of normal intensity. She lay in bed tossing about rather restlessly. Her mood was one of a whining depression, with some irritability, but no apprehensiveness. She looked somewhat dull, and her attention could at times be attracted only with marked difficulty, again quite readily; but we were struck with the fact that now and then, even at the time when we had difficulty in obtaining answers, she made occasional comments on things which were said in her hearing. Hallucinations were at times quite prominent; she had spoken of hearing bells ringing, had seen pictures on the door, her sister in the pillow, a man in her bed, and she tried to pick imaginary flies from the bed-clothes. She was completely disoriented; though she repeatedly called the physician "doctor," the nurse "nurse," yet again she miscalled them. Paraphasic turns of speech were quite marked, as we shall presently show. For the two succeeding weeks her condition remained essentially unchanged, and

may be summarized as follows: Sometimes she appeared dull, even to the extent of soiling herself. Her attention varied: it either could be easily attracted or this was very difficult, and she could be pricked with a pin without any reaction. Her disorientation remained, though shaded off gradually. She thought she was in New York and other places; again, called people by wrong names. Her time orientation was very poor. Sometimes she related delirious experiences; for example, said that she had been up the river lately in a boat, or that she had just been in the woods, where "some money was tied to a tree," and the like. The hallucinations continued, and even became more marked. She heard voices, reached out her hands to fancied visitors, talked to the wall, spoke of the girls upstairs "who have talked" about her, and quite marked was the fact that she picked up imaginary threads from the bed-clothes. Artificial hallucinations could be produced by rubbing her eyes. On such occasions she said she saw "a fire-place," "woodwork," "shelves," "a woman in a blue dress," "all sorts of things." Reading tests showed fair results at times; again, she made glaring mistakes, such as reading "pollies" instead of "1903." When questioned about events in her life she varied a good deal, sometimes gave perfectly absurd answers, e. g., that she was born in 1881; again, the answers were apparently perfectly correct. A few tests to study her ability to retain impressions (Merkfähigkeit) yielded results which would make one think that this was very poor; but the question of attention was not sufficiently considered at the time, so that we must not lay too much stress on the results, all the more so since it was found repeatedly that at the end of an examination she remembered incidents which had taken place at the beginning of it. Her talk may be illustrated by the following examples. She said spontaneously: "I'll never see my mother any more; she has been trying to hold up since she was lost." And then, pointing to the nurse, she said, "This is my mother. Please let me go. There is nothing for me to stay here. That's what I was, freezing. It seems just like she came in the window." (What do you mean?) "Well, don't you know there is a store in front of the bridge that comes right down to a point of lace. She lived there, or she did when I lived there," etc. "Down to a point of lace" is evidently a paraphasic turn, a trait which may be further illuustrated by the following samples. "We were coming down the ref road . . . I can't tell you where it is, it's the mostly jardmar, in the mell, mell jar, in the worsted mill yard." Or in speaking of Chattanooga, she said, "Chattanulgo, Challamutta"; and on one occasion when she heard a telephone ringing, she said it was the Chattanooga ringing, or "You are the gentleman I not in the grocery store."

In general it may be said, as is the case in these patients, that though the talk showed some shifting of subjects, loosely connected, it was not that which made it difficult to follow it; nor was this a very marked trait, as she kept often to the subject she had chosen fairly well; but it was the fact that she told of delirious experiences which we knew nothing about, and the talk was further obscured by the paraphasia.

After the two weeks the patient gradually became perfectly clear, orientation was excellent, the hallucinations disappeared, and she talked very naturally. It was all the more striking that with this clearness she retained for a number of days a belief in some of her delirious experiences, without however showing an adequate affective reaction. Thus she claimed that the nurse had told her that she had killed a man, and said she knew it was her husband. She explained that at home her husband discharged a nurse, and that the latter followed him to the barber shop and shot him through the thumb. When questioned retrospectively about the events which had occurred in the hospital, it was found that the very first part was practically a blank to her, but that after that she remembered quite a number of things, which, however, were not put together in anything like a sequence. She was taken home before she had entirely ceased to believe in some of her delirious experiences, although she did not at all react to them.

Case 2.—Mrs. W., aged 30. In the hospital from May 23 to August 3, 1903. Her maternal grandmother was insane for fifteen years until her death at the age of 60; her mother had repeated attacks of "nervous prostration," and one of the mother's brothers was an epileptic. A paternal uncle had an attack of insanity.

The patient herself had "nervous prostration" when 22, a condition in which she complained of considerable physical weakness, also of much pain in head and spine, and is said to have been very "hypochondriacal." She was in bed for months. She was married two years after the onset, but only two years later, i. e., four from the beginning, was she considered really well.

Three months before admission the patient is said to have had an attack of "grippe." She was weak after it, complained of palpitation, and was considerably worried about it. She had to remain in bed, became nervous and irritable, and more and more worried about her condition. It is claimed that she would sometimes stare for half an hour at a time. A month before admission she attempted suicide for the first time, and was henceforth very insistent in her attempts. Three weeks before admission there were occasional spells of mental clouding, and for a week before admission she had been rambling, noisy, resistive; finally confused, untidy, hallucinating, eating almost nothing for some days.

Fortunately we have a good account of the drugs which this patient received. It must be remembered that she was admitted on the twenty-third of May. From April 1 to 11 she was given 10 grains of bromide at night. From the eleventh until the twenty-seventh it was replaced by 18 grains of trional, repeated if necessary. From April 26 to May 3 she had 60 drops of Tr. hyoscyamus a day. From May 3 until admission she had regularly, at first 60, then 120 grains of bromide, plus 15 drops of Tr. gelsemium a day. In addition to that she had, for the week preceding admission, altogether 2½ grains of morphine and $\frac{1}{100}$ of hyoscine. And finally she was given Tr. passiflora, 5 to 10 drops, every 2 to 3 hours; later, 30 drops at longer intervals.

The patient was admitted with a temperature of 100° F., sallow appearance, foul breath, heavily coated tongue, pulse 100. She was restless, shouted for her husband, spoke of hearing her people murdered, of seeing coffins, men with revolvers. She frequently seemed to pick up things from the bedclothes, and when questioned said she saw bugs and threads. Her voice was thick and her stream of talk fragmentary. She was completely disorientated. At the morning visit her physical state was the same as described. In addition it was found that there was no tremor, but marked exaggeration of reflexes, with pronounced ankle clonus, inexhaustible on the right side, exhausted after 10 to 12 motions on the left. Babinski absent. She lay in bed quietly, mumbling something to herself, occasionally calling out, evidently in response to hallucinations, sometimes picking imaginary things from the bedclothes. She appeared dull. The mood was indifferent, there was neither fear nor any evident depression or exhilaration. It was sometimes very difficult to attract her attention, again more easy. Sometimes she commented on slight, quite unobtusive noises, such as a distant train. Orientation was poor. She said she did not know where she was, did not know the people, but she gave the month as May, the year as 1903, then 1902. Again, she said she was at her sister's house, but frequently called the doctor "doctor," the nurse "nurse."

Her talk may be illustrated by the following. When asked how long she had been sick, she said, "I have been sick eight or ten weeks—that is if I speak right—now my folks tried to lose me, they were hunting for me." (Did you see them?) "I could not hear a sound, only her [nurse], and she will kill me" (no affect). "They all say I was afraid because I went to a store on Tremont Ave. They would not let me have—well, she would not let me—have anything to do—you remember that [to nurse]—she can't find out. I'm growing hazier and hazier—but this forenoon, well, I'll tell you what she did. I see her object in it now. I hadn't thought of it. I have been moved so often. We have moved around in the daytime—in the night—we have moved all around, I don't know how many things," etc.

What is not brought out in this sample is her paraphasic turns, which, nevertheless, were quite marked. Thus she said, in good connection, "That is all the satisfaction I can get, and I am satisfaction." Or when asked the day, she said, "I don't know, I haven't seen a map for ages. I am just 8:30 May something." Or again, "Are you the gentleman that's marrying this house?" Or, "He make it distinct enough that I would not get well. Distinct, extinct enough, he made it excitement enough," etc.

When asked memory questions she varied, evidently owing to her variation in responsiveness. She gave her age correctly. (Have you a child?) "Yes, three years ago" (correct). (Is the child living?) "No, dead" (incorrect). (How long ago since it died?) "Two years." (What did it die of?) "Still-birth." (What?) "Two years ago the 8th of February." (What happened then?) "A boy was born to me."

(How long did he live?) "Oh. I was taken sick on the eighth and he was born on the ninth." (Is the boy living now?) "Yes." (What is his name?) She gave it correctly. (Have you ever lost a child?) "No" (correct). (How old is your boy?) "33." (No, your boy?) "3" (correct). Then she was asked, "What is 9 times 15?" She said 19. (7 × 13?) "21." (8 × 9?) "72." (16 × 12?) "72." (What is the capital of the U. S.?) "Boston." (Capital of Maine?) "45." (Capital of Maine?) "Capital of Maine? 75." (What is 75?) "A number." Then she was again asked impressively, and she said correctly, "Augusta."

In addition to the hallucinations above described, artificial hallcinations could be produced by rubbing her eyes. She said she saw "a horse-car on the street," "a post," "a white post," "people and a dog." (What kind of people?) "Mostly Chinese women." (What color of dresses?) "Mostly white dresses. I saw a cap just now—all kinds of things, houses and everything else." When told to open the eyes she said, "Now I see a bunch of grapes." Asked what she saw on the ceiling, she said, "Grapes—single grapes, small and large ones."

Just as we found in the other cases, this woman made striking mistakes in her reading.

This condition lasted about ten days, while the more marked symptoms gradually faded, the tongue became clean, the reflexes normal, the orientation became much better, the paraphasia was slight, the talk was much clearer, but in spite of all this improvement she continued to believe in the delirious experiences and for a time hallucinations persisted. Although she finally cleared up altogether, she held on to some delirious experiences almost to the end, while at the same time she showed a certain mental sluggishness.

Case 3.—Mrs. E., age, 43. In the hospital from July 22 to September 15, 1904. Heredity is denied, and the patient has never before been insane. A year before admission she had a good deal of worry. She lost flesh and got weak, slept poorly, and it is stated that at that time she took a considerable amount of morphine, but that she had not taken any for three months. For about three weeks she has felt very exhausted, slept poorly, complained of many pains, and it is stated that a great many drugs were then given her, but we were unable to find out just what. She got steadily worse, finally somewhat confused, and three weeks before admission she was sent to Boston. There she had to be looked after, had to be dressed, fed, and gradually became excited and at times fearful, confused, so that twelve days before admission she was taken to a small hospital, where she was dull, untidy, restless, had hallucinations of hearing and vision.

In this hospital she was again given hypnotics, but as has often been our experience, the guilty physicians are very apt to be exceedingly general in their answers to letters of inquiry about drugs.

The patient was brought to us in a state of marked dulness and hebetude; she showed a tendency to keep her eyes closed, was untidy, her mouth was dry, presented sordes, the tongue was heavily coated, the

breath foul, the pulse rather weak (100). The internal organs presented no abnormality. The reflexes were normal. There was no terror. She lay muttering, speaking indistinctly and thickly, but when her attention was attracted her talk was much more connected and the voice much less thick. Sometimes it was easy to attract her attention; again, difficult. But it was quite striking that she repeatedly caught up statements made within hearing and commented on them. Her train of thought was at times difficult to follow, partly on account of paraphasic utterances, partly because she spoke of things irrelevant to the situation. But she kept on the chosen subject remarkably well. The answers were often quite irrelevant, evidently because she either paid no attention to the question or because of her paraphasic turns. We may give a few examples. When asked what is two times two, she said "two over"; and again, asked what's two times two, 'that what I said, you would think I was crazy, a woman of 75 to make me marry" (she had spoken of that before), "to be asked why I did not marry such a woman" (paraphasia). Then turning to the nurse: "Florence. No, that isn't Florence. I said 'put that feather, over there,' and Florence said, 'No, I won't put that feather over,'" etc.

The data of her life were at times given well, again poorly. She was totally disoriented, miscalled people. Even simple multiplications were done poorly. Her mood was either indifferent or somewhat euphoric. Hallucinations were present and frequent, especially those of hearing, and to a lesser degree those of sight. Quite striking were the tactile hallucinations, or tactile and visual combined, which were manifested by her imaginary picking up objects. Her ability to retain impressions tested in the ordinary way (given a number of 4 digits to remember) appeared poor, but here again we must add that such a test is only of value if the mental responsiveness is taken into account. Paraphasia was pronounced. Interesting were the results when objects were shown to her. They were evidently in part due to a disorder in apprehension, so clearly brought out in Case 4, and quite striking was also the influence perseveration. The following samples may be mentioned. (Knife.) "Brick house." (Knife.) "Those are—" (thinking). (Tell me.) "Knife." (Bunch of keys.) "A key ringer—ringer for keys." (Watch.) "Keys." (Charm.) "A charm." (Spectacles.) "Those rings which go on." (Cuff.) "Keys, cuff of keys with a key-note in it." (Pink.) "Pink." (Palm leaf fan.) "Fan." (Brown book.) "Bible." (Hand-glass.) "Looking-glass." (Comb.) "I don't know—that's my black comb." (Hair-brush.) "Comb. It's a hair brush." (Closed fan.) "A fan, a parasol, a very little parasol." (Opened fan.) "A fan, a parasol." (Cuff button.) "A gold ring." (50 cents.) "A quarter." (25 cents.) "A quarter." (50 cents.) "A quarter." (5 cents.) "10 cents." (One cent.) "5 cents."

Three days after entrance the attention was attracted with greater ease, but the paraphasia persisted to a marked degree. She read very poorly. For example, when made to read "bats have proportionately the

longest ears and the oddest shaped noses in the whole animal kingdom," she read "Bates properly continue the largest earnestly and clearly noses of the kind, of the innumerable kind." When she was shown pictures she showed marked abnormalities, pointed out birds where there were none, called a piece of bread in the hand of a little child "a cucumber squash," saw "a lobster claw" on a piece of paper which contained only indistinct marks, not at all suggestive of a lobster claw to a normal person, or she caller three lambs "three cans" (paraphasic?). At that time she was still disoriented as to place. In regard to time she knew the month and year, but nothing more. She miscalled persons, but not consistently. She gave no one a correct name, but called the doctor "doctor," the nurse "nurse." The hallucinations continued. She heard voices, and still picked imaginary things from her bed-clothes. In regard to the disorientation, it may be mentioned that she thought she was at home, or in the house of a friend. She repeatedly told of delirious experiences.

In a few more days the hallucinations left, she became perfectly clear and the attention was good, but she still called the place wrongly, still uttered delirious experiences. Thus she told of an accident which had happened in which her mother had been injured, and claimed that the examining physicain had been called in and had operated on the mother at her home. Gradually she cleared up entirely, not only from her delirium, but from the condition which had originally led to the giving of drugs.

Case 4.—Amelia G., aged 39. Dressmaker. Admitted January 11, 1905.

The patient has some psychopathic heredity, and it is said that she was always of a suspicious nature, was easily frightened, and inclined to be quite hypochondriacal in the sense of making a great deal of small ailments. For ten years she complained much of pain in the neck and head, but on the whole was able to do her work.

Six weeks before admission she complained more of the pain, became depressed, despondent, listless; sometimes she was restless.

Five days before admission she became more depressed, self-accusatory, and sat for hours without speaking. Soon after this she began to "talk queerly," said people were dead, that she had killed six little children. She also said that the top of her head was "blown up." She claimed that her mind was gone. At the same time she showed indications of morbid self-reference, thought things which were done had a peculiar meaning, and she fancied that people looked at her. A few days before admission, hallucinations began; she answered voices, and she saw "red devils crawling over the sister's jacket," "a little angel walking round the rim of her drinking cup." She was often seen staring.

For about a week she had eaten very little and had slept very poorly.

Now, this woman had been given liberal doses of bromides in the six weeks preceding her admission to the hospital. We were unfortunately unable to find out the exact doses, but it is said that she was given a teaspoonful of a bromide mixture every three hours. The fact that at entrance she had marked acneform eruption on her body also supports the

supposition that she had been heavily dosed. Besides these bromides, she was given hypodermic injections, the nature of which we could not find out.

At entrance the patient showed, as was stated, an acneform eruption; the tongue had a heavy brown coat; her breath was foul. Her gait was somewhat unsteady, resembling that of cerebellar ataxia. But there was no tremor, the reflexes were normal. The pupils could not be tested on account of lack of co-operation. She showed marked tenderness and pain over the joints of the legs, but no swelling. Her urine showed a slight trace of albumin, but nothing pathological otherwise, except a very high specific gravity, .1041. Temperature normal. Pulse and respiration showed nothing of any consequence.

She wandered aimlessly about, presenting the uncertain movements above described. Her expression was strikingly empty, but not immobile. She made the impression of being absorbed in vague thoughts, and very often she did not answer questions, or what she did say had no bearing on what she had been asked, but was either a vague allusion to the "Blessed Virgin" or the like, or a repetition of something she happened to hear, and the result was the same whether complicated or the most simple questions were asked. But she showed her tongue, and reacted quickly to pin pricks. Quite striking was an aimless resistance, blind in character, yet without an affectful background to it, making rather the impression of a tendency to perseveration, a trait which was later on brought out more clearly. Interesting is the fact that with this there was at times a tendency to catalepsy, and above all a marked, though not consistent, echopraxia, even to tests. It should be noted that in spite of all these traits she at times occupied herself with the physician, fumbling aimlessly about his clothes and the like.

Next day the condition was quite different and remained different for about a week, after which time it very gradually shaded into a typical state of manic depressive retardation, which persisted so long as I observed her. The condition which developed on the second day, and which we shall presently describe, was a delirious state, and for some weeks after the height of it was passed the slight delirious traits persisted, masking the manic-depressive retardation, so that for quite a while the case presented considerable difficulties to the correct interpretation.

During the delirious condition she was at first completely disoriented as to place, persons, time, even the time of the day. Whether this had been so on the first day we were unable to decide, because she did not answer questions. It could be established, after the first day, because she responded more readily, although she had a marked tendency from time to time to get into a similar staring condition as at first, and even to become decidedly drowsy. These variations in her responsiveness were quite marked, so that at times it was impossible to attract her attention. When thus absorbed she did not react to pin pricks, and, at these times also, it was found that she would firmly hold on to anything which she happened to have in her hands, so that it could not be taken away from her except when it was possible by putting some other object in front of

her eyes to forcibly attract her attention to that. Again, when looking at anyone, she would follow that person with her eyes when he moved about. All this made the impression of a peculiar fascination and perseveration. During this time she lay in bed, often appearing rather dull. She hallucinated, saw "staggering things with long legs," "a bird" in the physician's hair, "lots of children at the end of the hall," or she saw faces in the transom, and heard voices. But she had evidently no hallucinations of touch. Her talk, which was rather scanty, was, however, clear, and there were only occasional paraphasic turns in it, but these were distinct. She produced, however, a number of delirious experiences. She said she had been "in a dry goods store this morning," that she had gone down a long street, and the like.

The mood during all this time was strikingly indifferent, even when she uttered occasional depressive ideas.

We then made some experiments daily in order to study more closely the hallucinations, the process of apprehension, and her ability to retain recent impressions. In all these experiments the question of mental responsiveness had to be taken into consideration, so that we also made some tests regarding this.[3]

Let us first consider the hallucinations. Like all the other cases, this patient showed marked artificial hallucinations, i. e., when the eyes were pressed upon and she was asked what she saw, she said, for example, "a whole pile of black iron rails"; later, "I see a little girl of 13 or 14 holding a doll." (What kind of dress has she on?) "A gray one." "I see a baby carriage." She also said, "I see a man," or again, "It looked like a yellow suit with brown buttons on it." As will be remembered, it was Liepmann who first showed that such hallucinations could be produced in alcoholic deliria.

When pictures were shown to her the halluciantions were also very marked, just as had been the case in Mrs. H. Thus in one picture which she first described quite well, she added, "and there is a man crawling under the fence." In another picture she pointed out a cat in the grass, where there was none. After having described the essentials of a third picture correctly, she added, pointing to rather small, indistinct geese, that they were birds. A small brown chicken she called a squirrel. Finally she saw "a big snake and a big green lizard." (The picture showed a patch of grass.) When she was shown a fourth picture she again described the essentials correctly, but when she came to an indistinct chick she said, "There is something here but I can't see it." Later she saw "bugs running up the shrubbery," and finally "a long green snake." In other words, the patient began invariably by describing the picture correctly. That was at a time when her attention was attracted by a new picture, but soon she began to hallucinate, and as we shall presently

[3] These experiments I made in conjunction with my friend and associate, Dr. S. I. Franz, to whom I wish here to extend my thanks for his assistance.

point out, she began to see indistinctly, and when one watched her further she was very apt to go off, as it were, i. e., to get into a staring state similar to the one described on the first day, or she got distinctly drowsy.

That she does not see well we infer from the fact that she pointed to the chick saying, "I cannot see that." However, this was rather isolated and usually she hallucinated. Some years ago I had occasion to observe a case of Korsakow's psychosis quite early in the course. This man resembled in many ways the patient under consideration. In that case it was very evident that he had periods when his vision was very indistinct. He also hallucinated at times during these periods of indistinctness of vision; more often this was not the case. The Korsakow case differed very markedly from Miss G. by making a much more natural impression, but from time to time he had peculiar short spells in which he seemed to wander, would not respond, and sometimes even his attention could not be attracted for the space of a minute or so. My attention was first called to this condition while I was making a sensory examination. He would answer promptly for a time, then suddenly he could be touched or pricked without making any response. In order to study this more carefully we applied the following tests. We read to him columns of thirty-two figures each, among which five threes were irregularly distributed. He was asked to tap the table every time he heard a three. He would often allow from one to five threes to pass unnoticed, on one occasion fourteen in four lines, and altogether 14 per cent. When this man was shown series of letters (we used quite large ones) or pictures, he would at times name or describe them very well. At other times he would say, "it's dull," or "it's blurred," or "it's going," or simply, "I can't see it." Although the most frequent result was that his vision became merely blurred, he, at times, hallucinated like Miss G. For example, on one occasion, instead of seeing a letter he said he saw "a procession of the Knights of Pythias." A few times he also had auditory hallucinations in such periods. Questioned about these states he said, "My mind wanders"; or again, "I get forgetful at those times."[4] We see, then, that this patient had short periods during which his "mind wandered." In these, his attention could at times not be attracted; at other times he showed a peculiar visual disorder, and with it a tendency to hallucinations. The analogy with the case of Miss G. is obvious. The most likely cause of this visual disorder seems to be a disorder of accommodation and fixation. There can be no doubt but that this indistinctness of vision plays a part in the production of the visual hallucinations, or more correctly, illusions. The most important part, however, we must admit to be the

[4] An interesting feature about the case was quite marked variation in the blood-pressure, distinctly perceived by the touch. But I was never sufficiently satisfied to declare that they were synchronous with these periods. On one occasion Dr. Amadon established the fact that the fundus, which in the beginning of the ophthalmoscopic examination appeared normal, later was much paler.

mental alteration, namely, the peculiar dipping down to lower levels of consciousness—if this term may be permitted—a condition of mental dissociation analogous to dreaming or to the hypnagogic state, in which hallucinations are also present. And we all know that in the state preceding sleep our vision becomes indistinct, as everyone has experienced when trying to read a book while having difficulty in keeping awake.

We will now return to the case of Miss G., and to the experiments on the process of apprehension. We wished to see whether a short exposure of letters or words or pictures was sufficient for her to apprehend correctly. We used for that purpose a small screen of a photographic apparatus, the exposure of which varied somewhat between one tenth and one fourth of a second. Among seventy tests we found that sometimes we obtained, even with the shortest exposures, remarkably good results, which in no way differed from the normal. This was especially the case with simple letters or with words. At other times the results were remarkably poor, and again the patient hallucinated. The influence of the clearness of the object was evidently of some importance. Thus, when an indistinct bird was shown, she said, "I see three cows in a field and a man coming along with a rake over his shoulder." Bonhoeffer, in studying his alcoholic deliria, has pointed out that by means of the æsthesiometer we sometimes get normal, again very bad results; in fact, his findings are perfectly analogous to ours. We may say that, from time to time, there is a most profound inability to apprehend, but that this is due entirely to the specific delirious alterations, the dipping down to lower levels of consciousness; while at other times we obtain normal results.

Somewhat more complicated is the study of the retentive faculty (Merkfähigkeit). When we gave the patient eight consecutive figures to repeat, she was able to give on an average about four; a few times, however, she gave seven and six, sometimes none or only one (nineteen tests). It is possible that seven and six represent her normal limit.

Other tests were the following. The patient was given pairs of words —(1) words connected by habitual association, such as "bread and butter"; (2) pairs of words connected by internal association, *e. g.*, "head —hair"; (3) pairs of words which were not connected at all, such as "screen—ball." After times varying from thirty seconds to two minutes, thirty minutes, an hour, or even one or two days, she was given the first word and had to supply the second. We found that she was unable to retain words which were not connected, but we made few experiments with these. Among the words with internal connection she retained 31 per cent.; among those with habitual association 57 per cent. It was generally found that when she was able to retain the words for thirty seconds she also could retain them for much longer periods, and the results with habitual associations were even strikingly good when she was asked two or three days afterwards.[5] In this connection we may also mention some experiments with pictures. Three days after she had been shown certain

[5] These word-pair experiments were made with twelve different word-pairs on five different occasions.

pictures she was able to pick out correctly the five shown among twelve. And similar evidence of her ability to retain impressions was seen from day to day when questions about incidents of former interviews were asked. I doubt whether the results would have been the same in alcoholic delirium, for which Bonhoeffer claims such a memory defect, although he is not very explicit about it. At any rate, in view of these results, it seems very questionable whether we can speak in this case of a memory defect independent of the general clouding of consciousness. It might very well be that in alcoholic deliria, which have many points of relation with Korsakow's disease, there exists an independent memory defect, while this is not true in cases here under consideration. Finally, experiments similar to those recorded in relation with the Korsakow case above mentioned were made, *i. e.,* the patient had to tap every time a three occurred in a column of figures read to her. She omitted 34 per cent. These tests were made at two different periods—(1) when the delirious traits were more in the foreground; (2) when the retardation was more pronounced. During the former there were present 16.5 per cent. omissions, and 4.8 per cent. slow reactions; during the latter, 51.5 per cent. omissions, and 4.8 per cent. slow reactions.

If we summarise the clinical picture of these drug deliria, we find in the first place on the physical side invariably a coated tongue, a foul breath, sordes at times. We also find occasional slight febrile movements, sometimes unsteadiness of the gait, increase of reflexes, and some slight, but quite inconstant, tremor, The speech defect I am inclined to attribute in part to the bad condition of the mouth, in part to the clouding of consciousness, because it is very striking how much better these patients speak when they are aroused. There is no cyanosis and no flushing; on the contrary, the complexion of these patients appears rather pasty.

On the mental side we find first of all a certain dulness and hebetude, so that it is at times difficult to arouse these patients, while at the same interview it may be quite easy; and we have repeatedly noted that in spite of a marked dulness, unobtrusive noises may be commented upon. In harmony with this dulness is the fact that we often find a certain drowsiness even in the mildest cases. We shall later return to this.

The most marked alteration is a constant tendency to dip down to a lower level of consciousness. This seems to me a more correct formulation than to speak of an attention disorder, which term is used, for example, for the very different alteration underlying flight of ideas; although it is to be expected, and

experience actually teaches us, that the lowering of consciousness which we here speak of should lead to an attention disorder, as a partial secondary manifestation, which then, of course, presents itself in a very different setting than that which produces a flight of ideas. When the consciousness sinks to this lower level we have a condition somewhat akin to sleep, inasmuch as there is a general dissociation; spontaneous trains of thought arise, not connected with the outside world or with reality, very similar to dreams. At the same time there are hallucinations of various senses, more especially sight, hearing, and touch. These hallucinations may be produced artificially by rubbing the eyes; they are also well observed if the patient is made to describe pictures or to read. We have seen that the visual hallucinations, or better the visual illusions, are in part at least due to an indistinctness of vision which we have reason to attribute to insufficient accommodation and fixation. However, the essential factor in the production of these hallucinations is evidently the general dissociation for which we find an analogy in the hypnagogic hallucinations and in dreams, and indeed it seems not improbable that hallucinations are most frequently produced by a dissociation of some kind or other.

It should again be emphasized that this, we might almost say, specific delirious tendency to dip down to a lower level of consciousness, is but a tendency, and that the patient can usually be roused, often to strikingly good, connected activity, as was shown in all our patients, especially well in the tests applied in the case of Miss G. The paraphasia seems entirely due to the lack of attention, the inability to concentrated activity as the result of the specific delirious alteration, as Bonhoeffer has shown.

The disorientation must also be explained on the ground of this delirious change, and we have seen that in these drug cases a memory defect, independent of the specific alteration, can probably not be made responsible for this disorientation. But one thing should be mentioned in this connection. We have been struck with the fact that delirious experiences and delirious interpretations are held with remarkable tenacity, even during the convalescent stage, at a time when the patient is otherwise perfectly clear, and it is not improbable that this peculiar ten-

dency, from an explanation of which we would refrain, is to a great extent responsible for the lack of correction which one would naturally expect in such patients who from time to time can be aroused to a connected mental activity.

As we have stated, the retentive faculty, or the memory for recent events as such, is probably not altered independently, and the same may be said in regard to the memory for old events.

The train of thought shows some characteristics which resemble those of flight of ideas, and are due, as we have said, to the incidental attention disorder, while at other times the connection is retained for considerable periods of time. What makes the utterances of the patient at times so incomprehensible to us is not this tendency to flighty turns, but rather the fact that delirious experiences are related with which we are not acquainted, and it is further made incomprehensible by the very frequent paraphasic elements.

The mood is often indifferent, but we have seen in one case a certain euphoria, again a certain whining depression, some indications of apprehensiveness, *but never fear.*

So far as the motor side is concerned, we may find a certain restlessness or disinclination to move, but all this seems incidental to the essential delirious alteration: as a rule it shows nothing very pronounced.

We will finally compare with this picture that of the alcoholic delirium as Bonhoeffer describes it. According to this writer, this psychosis presents in 80 to 90 per cent. of the cases the following characteristics. The patient moves about a good deal, and is constantly occupied. His face is congested, his expression anxious. Often he shows marked fear. There is a very pronounced tremor, profuse perspiration. The gait may be somewhat uncertain, and there is ataxia of speech. We may add here that he mentions occasional eye muscle disorders, which are, however, slight; and, retrospectively, the patients may speak of double vision.

The patients do not appear dull, and even at the height of the delirium they can be demonstrated to students, and the impression made on them is that the patient's manner of reaction is not markedly different from the normal; but the examiner finds that it takes some effort to hold the patient's attention. On

a more careful examination, Bonhoeffer established the following. It is possible at any time to force the patient to a maximum degree of attention which does not differ from the normal. This may be shown, for example, by experiments with the æsthesiometer. A conversation with the patient also tends decidedly to raise his attention to a certain level, but when he is left to himself there is a constant tendency for the attention to reach a lower level, at which time the normal train of thought ceases, and the arising ideas show a marked tendency to become projected, as it were, as hallucinations. During an examination, when the attention is raised to a higher level, hallucinations are very few or totally absent, and the diminished attention shows itself chiefly by signs which are very similar to those of a normal inattentive state, such as a paraphasia similar to the fatigue paraphasia.

The memory for old events is not interfered with, and simple calculations are done well, as are all habitual tasks; but where a concentration is needed, and combinatory efforts are required, the patient fails. The retentive faculty, however, is markedly altered. On the ground of these deviations, Bonhoeffer explains the disorientation which in these cases is very marked. He also mentions in this connection a decided suggestibility and a marked tendency to confabulation, which we all know so well from our experience with Korsakow cases. From these confabulations he justly separates those which arise from hallucinations.

Bonhoeffer devotes considerable space to the hallucinations. He raises the question whether central or peripheral causes give rise to them. Meynert has claimed that in deliria the projection systems were at fault, and others had found various disorders, such as amblyopias (Magnan), retracted field of vision (Kruckenberg), disturbance of colour sensibility (Galezowsky). But Bonhoeffer points out how, on careful examination, he was unable to find any of these changes, except perhaps in colour vision He is of the opinion that peripheral changes, if they are of any consequence at all, have to be given a very subordinate place in the production of hallucinations. He mentions casually Mendel's claim that disorders of accommodation have something to do with visual hallucinations, but he takes no position in the matter. In describing the many mistakes which such patients make in read-

ing, he says, however, that possibly the difficulty of convergence may partly cause this disorder, since he obtained better reading with monocular vision. He points out the well-known fact that the hallucinations in delirium tremens are apt to be combined, so that entire scenes are hallucinated; and he emphasises the frequency of the illusionary character of hallucinations, which are, after all, frequently a projection of the patient's thoughts. Just as Liepmann, so Bonhoeffer found artificial hallucinations produced by pressure on the eyeball, and hallucinations were also produced by looking at pictures, or by the reading tests.

Now the deviations from this picture are found either in complications with other psychoses or with epilepsy; but what interests us here especially is his description of the more severe cases. Such patients are more difficult to fix; finer tests cannot be applied. They are duller. The motor excitement is coarser, more elementary, the cyanosis is more marked, sweating and anxiety greater, the speech like that in meningitis. Eye muscle palsies are more frequent, as are various other paralytic phenomena. Such cases are very apt to terminate fatally.

If we now compare the two pictures, that of our deliria, and that of the alcoholic delirium as described by Bonhoeffer, we find, in the first place, that that which we have called the specific delirious alteration is present in both. The hallucinations are the same, and here, as well as there, it is easy to produce artificially these hallucinations; they are seen when pictures are described, and the results of the reading tests, *e. g.,* are practically identical.

But all this we only find by a careful analysis, whereas superficially the two states differ so much that one would never be inclined to mistake the one for the other. That is due, in the first place, to the fact that we find in the alcoholic delirium the dilatation of peripheral vessels, and a tendency to cyanosis, and often evidence of anxiety or fear. The pulse shows more marked alterations in alcoholic deliria. I am inclined to attribute these differences to the fact that the alcoholic delirium attacks persons who are chronic alcoholics, and whose cardio-vascular system, therefore, shows marked degenerative changes.

A further difference is to be found in the tremor, which is very marked in the alcoholic states, slight and inconstant in the drug deliria.

Above all, however, the general responsiveness of the patient is different. We have seen that, according to Bonhoeffer, the alcoholics do not appear dull, and often make a strikingly natural impression on a casual observer so far as their manner of reaction to questions is concerned. In contra-distinction to this, we find our patients presenting a certain dulness and hebetude, and it is much more difficult to rouse them than it is to rouse alcoholic patients. It was a very natural supposition to think that possibly this greater dulness might be due to a disorder of apprehension which was added to the delirious alteration, and it was for that reason that the experiments on apprehension were made. They showed us that this is not the case. One might, perhaps, say that we happened to see graver states, conditions of unusually great intensity, and that the more marked conditions of alcoholic deliria, such as Bonhoeffer describes, are quite analogous, but are fatal only for the reason that the cardio-vascular apparatus is weak in the alcoholic conditions. That this explanation is not sufficient, is shown by the marked tendency to drowsiness even in our mildest case, Miss G. Therefore it cannot be merely a question of intensity, but this hebetude seems to be a special feature of these deliria. For some reason or other it seems that although a high level of consciousness can be reached in both kinds of cases, the tendency to sink to lower levels is greater in the drug than in the alcoholic deliria.

To a certain extent the fact that the alcoholic patient is constantly busy may depend upon this same difference. Whether there is, in the alcoholic states, also a certain elementary motor excitability, I am unable to say.

We have above mentioned the fact that Bonhoeffer assumes the existence of a memory defect for recent events in alcoholic deliria. Our experiments in the drug deliria, although they perhaps do not allow a general conclusion, speak against such an assumption for our cases. And we have also stated that it would not be improbable that alcoholic conditions should present such a change though it be absent in our cases, because we know how often alcoholic deliria run into conditions of Korsakow's psychosis.

There is another symptom which Bonhoeffer mentions, the nature of which is as yet uncertain, viz. the great tendency to

confabulation which he found in the alcoholic deliria. The "confabulation" which occurred in our cases appeared to be due entirely to the spontaneous trains of thought which were analogous to dreams, and which in part were externalised as hallucinations. We have, therefore, throughout our descriptions, spoken of the patients "relating delirious experiences." The fact that defects in the retentive faculty seem to have some relation to true confabulation, would suggest the possibility that the absence of confabulation and the absence of a defect of this nature were related; and, conversely, the lack of confabulation might be used as an additional support for the claim that the retentive faculty is not interfered with.

We see, then, that although superficially the alcoholic and the drug deliria are so different that the casual observer would never be reminded of the one by looking at the other, they have nevertheless both the same nucleus, *i. e.,* the specific delirious alteration, which is only marked by certain special features characteristic of one or the other.

# REMARKS ON HABIT-DISORGANIZATIONS IN THE ESSENTIAL DETERIORATIONS, AND THE RELATION OF DETERIORATION TO THE PSYCHASTHENIC, NEURASTHENIC, HYSTERICAL AND OTHER CONSTITUTIONS[1]

By Adolf Meyer, M.D.

PROFESSOR OF PSYCHIATRY, JOHNS HOPKINS UNIVERSITY

For years I have been struck with the frequency, not to say uniformity, with which a number of peculiarities of make-up present themselves in the history of the cases which form the nucleus of the disease-group which deserves the name "essential deterioration process," or "dementia præcox," in the sense now generally accepted by most alienists and perfectly intelligible to those who prefer not to commit themselves to any special nosology. I refer to those cases of deterioration in whom we cannot point to any satisfactorily determined and experimentally or clinically demonstrable constellation of outside factors, as we do in alcoholic insanity or in general paralysis. The prototype of the disorder would be those patients who, without any special positive manifestations, undergo an apathetic deterioration. Many cases have some positive symptoms in the form of hypochondriacal additions, or paranoic developments with more or less deficient systematization, or some acute mental disturbance of a more or less characteristic type. The term *primary* dementia is avoided and replaced by essential dementia, because it has been used in a very promiscuous way so as to include also stuporous disorders whether they belong to this dementia group at all or not.

The main-spring of investigation is the question of establishing the definite constellations which lead to any deviation under consideration, so that it may be better understood and avoided or corrected. The present formulæ are heredity and stress, or heredity and auto-intoxication, both pointing to matters which can be reached but indirectly and which do not seem to me to

[1] First public formulation of the synthetic conception of dementia præcox. January 3, 1905.

touch the working principles of the disease which we want to understand and modify. Heredity is an extremely important statistical fact, and it embraces very largely the excuses alienists have to offer for their inability to cope with certain things. In the field of action we are forced to decide what we can do in the face of heredity. Stress undoubtedly involves more of what is directly at issue, but it also is an expression of excuse concerning a feature of modern civilization which cannot be changed by the physician. Auto-intoxication is the happy word which has all the advantages of humoral pathology and expresses the principle of many of the methods of patching up the disordered organism. It sounds like an expression of accuracy, but there are no direct methods of demonstrating anything specific. It is always but part of the disorder and the setting of this disorder is what is to be established. Everything points to evidently rather complex constellations, and our aim must be to pick out those factors which actually do the work.

During the past years more and more dissatisfaction has developed with Kraepelin's notion of disease-process. Already in my review of his fifth edition in 1896 I criticised the arbitrariness of calling dementia præcox a disorder of metabolism, and Stanley Hall has lately given an expression to very sensible criticism in the following remarks: "The terms dementia præcox, insanity of youth, primary dementia, hebephrenia, and katatonia are all partial and none of them are entirely satisfactory. These troubles do not by any means invariably issue in dementia, but if all did, the processes that produced and preceded the lesions, and not their effect, should furnish a truly scientific principle of characterization and nomenclature. Hegel said of Schelling's absolute, as many since might have said of Hartmann's unconscious, that it was not a true philosophic principle, because "all cows look alike in the dark," meaning that a purely negative principle can never be an adequate explanation. We may say the same of dementia præcox, which faintly suggests the propriety of giving to general paresis, which almost always has a fatal termination, a designation like thanatic dementia, based upon this fact. Early mental death is a result of the morbid processes we have here to study, but so it is of others. With senile dementia both the facts and the propriety of the name are very different."

Instead of suggesting a disease-process which would be apt to befall any individual without special predisposition, and without necessarily any heredity, I should propose for discussion the concept of habit-disorders as suggestive of investigation of fact and of modifiable and accessible factors, also not necessarily dependent on heredity. I refer briefly to such cases as the following:

Patient born in 1874; was healthy, never robust; delicate and social as a child. He avoided boys and cruel sports, talked little; at about fourteen while his disposition was very forgiving he became somewhat cranky, went little with boys, rather avoided girls, became jealous of his younger sister and resented her resistance to being bossed. He went to church a great deal. From the age of seventeen he did not want the sister around when his friends called; once he maltreated a young lady visitor by shoving chairs against her so that she fainted. For some time the patient was occupied as a clerk, finally he gave up work, stayed out all night and slept all day. During a sickness, 1896–97, he was very good, asked his younger sister to pray for him so he wouldn't die, but on his recovery he was again very ugly. For about a year he stayed in a dark basement room with the curtains down so that nobody would see him loafing. He resented the bringing in of lights. About July, 1897, he threatened his sister with a razor; he often threw bundles at the mother and sister and often swore at them in the morning. He claimed that the sister told people in the street he was nutty. The patient admits masturbation from the age of nine or ten, up to the age of seventeen, when he fainted while playing hockey; he was carried home, remained unconscious twenty minutes; he claims not to have masturbated since then, but says he used to think about women because he thought this would diminish the nocturnal emission. He explained that his animosity against the sister increased since one day she and some girls surprised him hugging a friend of hers. He also did not want his sister to call at a certain place on account of immorality being committed there. He thinks he was commented on for being out of work. No evidence of hallucinations, although he thinks women called him names, a soft-headed imp, and nutty. Physicians found him depressed, shamefaced.

The first diagnosis was sexual neurasthenia, with a change of character at puberty and increasing eccentricity of later years and ideas of persecution and suspicion and irritability against family. Within about five weeks after admission the patient stopped his work at the bakery. He smelled a bad smell, he never had had it before and didn't know whence it came. He refused several meals, said he heard some one say that he was sent here for boarding and somebody whose name he doesn't want to tell called him an imp. In November he developed stupor in which he finally rolled up his eyeballs, became cataleptic, had to be fed. He showed infiltration of one pulmonary apex. He remained in a catatonic stupor for a number of months, and emerged from it with a paranoid form of dementia.

These notes taken from an old history do not show as good investigation as one might desire, but show the general trend of observation which has been repeated in many cases.

Another patient who is just now under observation and at present in stupor may serve as an instance:

*C. H., born 1883. Habit-disorganization. Typical catatonic deterioration at 21.*

*Family History.*—Negative, but the father of the patient, a night watchman, has been moderately alcoholic since early life, occasionally drinking to excess; premature gray hair runs in his family.

The patient's mother died at 53, of some obscure lung trouble with hæmorrhage just before death.

*Personal History.*—The patient was said to have developed normally during childhood, though she was looked upon as "a nervous child," easily startled and subject to bad dreams. She began school at seven, was smart, and applied herself well, but at the age of eleven she seemed to be *failing* and was thought to be *studying too hard;* she grew thin, seemed *nervous* and complained of *headaches.* When she was twelve years old her mother died, and the patient was then in such poor health that her sister kept her at home. She was then a very quiet girl, often complained of her head, and her sister says: "You could see there was something working on her." The patient had desired to become a school teacher—she admired teachers because they were neat and well dressed. This seems to have been merely a fancy which passed away after she left school.

Her father's third marriage was unhappy through alcoholism, the family was scattered and the patient lived with her sister. After she began to menstruate at fourteen she brightened up, had fewer headaches and seemed to be in better health. Her sister never suspected any disordered sexual habits, but the patient states that she *began to masturbate when nine* years old; and she has probably continued up to the present time (masturbation observed in the hospital); its effects and whether or not it had anything to do with the failure at school and the later difficulty over work, cannot be proved directly. The patient says however—"It spoiled all my youth and my life—I wasn't like other girls—I didn't want to go out anywhere."

At sixteen she went to work in a hammock factory, but the work was considered too hard for her; she thought the noise gave her headache, and she was afraid of the machinery. She had attacks of nose bleed and finally a diarrhea, so that after five months she gave up this work. Seven months later she took a position in a paper factory where she worked for a few months until it was burned down.

The patient was now seventeen years old and about this time she had some scalp disease; her *hair* fell out and a patch of *gray* appeared on one side of her head. This worried her intensely; she gave much attention to covering up the spot, and had the hair cut and dyed. She was ashamed to

go out, and when she had the offer of another position she declined it because she feared remarks would be made about her hair. After this she remained at home to look after her father's house. It is stated that she did this satisfactorily, but she complained of *headaches and sleepiness in the morning* and would lie in bed late; she was alone most of the time, had no companions, and her father worked at night and slept through the day. Her father says she was "a stuck-up girl" and "testy"—especially about her clothing, wishing for better than she could afford.

Her sister says the patient never showed any desire for the company of young men, and, in fact avoided them, and remarked that she would never marry as she had seen enough of married life (referring to her father's). The patient says, however, that when nineteen she allowed two young men to take liberties with her, but without complete connection as she feared pregnancy. She attended to her religious duties, but showed no excessive fervor.

During her twentieth year she was complaining of constipation and had a "creepy sensation" in her abdomen which she thought was due to a *tape worm*. She read quack pamphlets and bought medicine from drug stores; she developed *hemorrhoids,* became despondent over her condition, and when she was twenty-one (January, 1904) she went to the Polyclinic Hospital for *operation*. After the operation she complained that the nurses had neglected her and that her bowels were full of gas; she wished to be taken home to die. After ten days she was removed to her sister's house, where she was very sensitive to noises and wished to be quiet. She suffered from incontinence of the rectum for a while, but this has passed away. She did not again speak of the tape worm, but was very nervous if her bowels failed to move daily, and she also expressed the idea that her *rectum* was *closing up*. She grew very thin and much run down physically after the operation.

Two months after the operation she returned to her father's house, and with the help of a hired woman, undertook to do the housekeeping. During March and April she had a severe attack of tonsillitis. During the summer she was very quiet, and to the neighbors appeared always lonesome; no friends came in and she didn't care to go out; she suffered from *amenorrhea* after the operation—she *worried over* this and also her past *sexual misdeeds* (patient's statement). About this time she thought she would like to study again and be a bookkeeper; she bought paper and pens, put her desk in order for writing but nothing came of it. At her father's suggestion she used to ride on the cars or sit in the parks; she apologized for spending money and not working any. All of this time she was sleeping poorly, complained of her head and was despondent about herself. The neighbors say that her father scolded her about the housework and she often said "I am a good girl" or "I can do nothing right for him." The father denies that he scolded her, but says she got very cranky through the summer and without reason complained that he hurt her feelings or said: "You break my heart." Shortly before she left home she spoke differently to her father—"I have done you wrong, I spent

your money, you worked fifteen months for nothing, you have no good clothes," etc. She began to be *afraid* to stay alone *at night,* and a week before her admission *at a party* in her sister's house, she remarked—"*Everybody is looking at me,* I don't know why." A few days before she left home she said—"I have been sick a long time and thought I was going to die, but now I think *Tom* (brother) *is going to get sick and die.*" One morning she prepared breakfast, but would not eat because she had to pray for everybody first—later she ate breakfast. Then followed *queer acts.*

On October 1, 1904, she went to see the priest and that night she was heard by the neighbors praying. The next morning she went to church dressed untidily, left candles burning in her room and poured kerosene oil on the steps for holy water. That day she threw some things out the window because a curse had been put on her by her father and she didn't wish him to have anything. That night, October 2, she went at ten o'clock to where her father was at work, said she wished to see a priest and spoke of having neglected her church while she had been sick. Her father accompanied her part of the way home, where she later disarranged the furniture and other things, and at midnight left the house in her night-dress; she was found by a policeman kneeling in front of the church. She told the officer correctly where she lived and returned home with him praying all the while. Later, when an ambulance came to take her to Bellevue, she said—"I am a good girl—my mother is dead—it's all my father's fault."

At Bellevue she said—"*I hear angels telling me how to pray when I lose my thoughts*—sisters and nuns are around me here *to save and purify the world,*" etc. At times she *sang* and *rhymed, would jump up suddenly* and gesticulate wildly and then fall back on her pillow as if asleep; she admitted that she heard voices, and while in Bellevue she held her right *arm under* a stream of *hot water* and sustained a severe burn; she later explained that she did this in order *to save the world.*

She came to the State Hospital October 10, 1904. On the ward, during the bath and in bed she presented a state of beatitude, walked with her eyes closed, prayed and sang, with slow gestures and theatrical pathos, responded in a monotonous, unctuous manner—passed from smiles to tears, assisted little when cared for by the nurses, even wet and soiled her bed, and when asked why she did so replied: "Although I'm 22 years old I have been transformed from a big girl into a small baby." Why? "Well the Lord said I was *too pure to be a woman,* and in order to save the world I had to be *once more a baby.*"

Her arm was slightly resistive, remained elevated from ½ to 1 minute, then dropped quickly and relaxed. Her productions were a monotonous and empty religious jumble, slowly produced in speech and song or mere whispers with devotional gestures, closed eyes and a saintly sufferer's mien, interrupted by some queer acts. The following is a sample of her spontaneous utterances: "I believe in the Lord God—Father Almighty and St. Joseph—Sweet Virgin Mary pray for us—Almighty God, have mercy on me—forgive me my sins—grant me remission of all my sins (crossing

herself). . . . Divine Catholic world—now and forever—A-amen—glory be to the Father, Son and Holy Ghost, as it was in the beginning . . . Mary is the branch and Jesus the flower—A-amen—at least you, my friend, sweet Jesus, have pity on me (long pause) that he may lead us into a more pure, good, Irish—divine—Catholic world." She then folded and unfolded the counterpane, continued to whisper prayers, held her hand before her face and licked it with her tongue. She had a correct appreciation of things, touched the physician accidentally, and said—"Excuse me, doctor." She noticed the laughing outside and said: "I don't need them—I don't want them to be laughing at me out there—I want to be left alone to say my prayers in peace and quietness . . . now let me sleep in peace and quietness, for I am about exhausted."

What is the matter, have you any trouble? "I have been sick, I guess—yes—oh, for four years. . . . It came from my stomach, I had stomach trouble—I had bowel trouble—I had chest trouble—throat trouble—face trouble—teeth trouble—nose trouble—eye trouble—compound or complex for me—head trouble—brain trouble and all the quarrelling—father squabbling and scolding all the time—he sent me out for bug medicine, and may Almighty God give that medicine to the one who started this business—this devil's island." Are you sad? "I was sad but not now, I'm happy now." Are you afraid? "No more, I was dreadfully—of everything on earth—for 10 years, since my mother died." Afraid of what? "Everything, no one would look at me or talk to me—only about me—they said everything bad about me, that I was a bad girl, but I was pure." She made some reference to her gray hair, and when asked to explain, said: "Part of my hair is gray—that's what near made the world come to an end—they all laughed at me, talked about me, and even drew up a play about me called Devil's Island." She referred to the other people on the ward as "all the good, true souls—I don't know whether they are going to save me or whether I am going to save them."

All this shows numerous, *at times senseless moralizing and ecclesiastic reiterations with the undercurrent of salvation achieved or longed for—for herself and the world;* no true flight nor a dearth of ideas in the full enumeration of her troubles but again evidence of stereotypy of form, utterance in contrasts and reference to Devil's Island, quarrels at home, being laughed at, etc. "I had worms and the worms had me."—"I want to save everybody and everybody wants to save me."—"I went out for bug medicine and the bugs took my way—I lost my way."—"You're Mr. Tiffany"—an interpretation of Dr. M.'s identity; also, when asked her own name she replied: "Baby Chadwick of the whole world—Divine—Irish—Catholic world—Amen."

*Physical examination showed increased tendon reflexes; fine tremor* of the tongue, face and fingers. Pupils somewhat variable in size, but react normally; a bright red line appears when the skin is marked, and once when excited (screaming) her whole body flushed deeply; feet and hands cool and moist. Pulse high (110–120), small and soft at the wrist; a large burn on the right arm; slight retroversion of the uterus; masturbation

observed. Subjective sensations of pins and needles through her body, "Catholic electricity," and a feeling as if she were "in power."

The first night in the hospital she was sleepless—singing, praying, taking off her night dress, kneeling on the floor. Once she assaulted a nurse, jumped out of bed, ran to the window, shouting: "Mr. Sullivan, Mr. Sullivan, save me, save me, I'm lost." The following morning her explanation of this episode was as follows: "Everything went wrong last night, good, pure, true and holy doctor—I led you astray," etc. A similar outbreak of excitement occurred during the physical examination and again the following night. After this she slept and claimed to feel better. She had put her arm under the faucet to save the world—"Good, kind, Catholic doctor, I gave up my whole right arm." She then claimed the nurse was the whole cause of the trouble and was irritable towards her. She later excused her actions by —"I was sick in my head, dizziness in my head—excuse me, doctor, I was out of my head and knew not what I was doing."

She had an attack of diarrhea just after admission, which subsided in four days, and her pulse also declined gradually to about 90.

Examination nine days after admission found her quieter, that is, with less spontaneity in productions, but she was still in an ecstatic, dreamlike but not dreamy state. She gave vague accounts of hallucinations, of "thoughts" from God and of commands to scald her arm, of being "in power" (pins and needles); she explained that she had called herself "Baby Chadwick" because that's what they had pinned on her blue dress in Bellevue. She was correctly oriented as to place and nearly so as to time. Still called Dr. M. "Mr. T," though she could give his name correctly. She showed a remarkably clear grasp on the remote past, but her statements in regard to more recent events were colored by her peculiar trend, *e. g.*, in regard to her admission she spoke of being taken to the "sanctuary," "where my bowels moved and my water passed from me—I call it that because I suppose Jesus did the same things I did." She spoke of the bath, as where she had again been baptized. Her attention was variable, but retention was good. A newspaper account of a railroad accident was correctly read, but then queerly elaborated—"It ran over my arm," etc. She gave the Lord's prayer, repeatedly making an omission which she did not appreciate. Calculation was erratic, at times giving random replies.

Her own description of her condition was very striking, *e. g.*, when asked if she was sick she replied: "I was, but not now, my head was bad."

Were you out of your head? "No, doctor, I had all my senses and faculties, but I was senseless." When asked why she had spat at the physician she again spoke of being "in power," and said: "There is a power over us and amongst all of us—a holy power—it may have been an electric power—I had all my senses and wasn't out of my head—it was a derangement of the mind that came over me." When asked to explain this said: "Well, it seems as if the world was coming to an end—the 20th century coming or some turn."

At the end of two weeks she rarely spoke spontaneously, but lay quietly in bed, showed some waxy rigidity in the limbs, kept given positions and was slow and constrained in all her movements. She gave strange accounting for her actions, *e. g.*, during an interview she would rise slowly from her chair, explaining that she did so "to be with the nurse"—"to try to be good," etc.

Why have you been standing? "To try to live."

Why do you hold up your arm? "For peace."

At the end of a month she had passed into a pseudo-stuporous state, was more rigid and resistive, did not always respond, though tears would often roll down her cheeks. She held saliva and required spoon feeding. She brightened up a little at the beginning of a course of catharsis but soon relapsed. Occasionally she was restless at night and once when asked where she wished to go, said: "To fight for my life—fight—fight—out—out—out away from here . . . follow me—follow me—I am going to serve the American Army and Navy with my life."

During 1905 the patient still maintained constrained attitudes; she sat the entire day with her hands on her knees, her head bent somewhat forward and tilted to one side; she rarely moved unless urged to do so; she was profoundly apathetic and careless about her personal habits. As a rule she answered very few questions and then gave mostly stereotyped pious phrases. Often tears were seen streaming down her cheeks. She held saliva in her mouth, sometimes refused food, again ate ravenously. Frequently she would burst out laughing or smile without any cause.

Since 1906 her behavior has shown little change; as a rule she is mute, lacks all initiative and is untidy in her habits. There is still a tendency to maintain given positions, and slight muscular stiffness is encountered in the limbs.

In other words we have here again a patient with precocious sexual instincts, inefficiency at school, and later in factory work, neurasthenoid symptom—complex with head and intestinal symptoms, growing seclusiveness, marked disorder of intestinal habits, amenorrhea, and finally development of a peculiar tantrum. Without any of the causes which are sufficient to bring about a similar dream-like phantastic state, in a form quite different from a delirium, or manic-depressive attack, or anxiety psychosis, or hysterical dream state, in a condition which was compatible with normal orientation and grasp on the environment, an odd state of longing for an enjoyment of salvation with a strange mystic-allegoric trend of action, interpretation and notions developed, probably most like a hysterical dream-state, but with much more deficiency in corrigible foundation and relation to things as they are. Occasionally there are acts of perplexity; the patient claims to hear

angels telling her to pray "when she loses her thoughts"; strange sensations make her feel "in power," telephone-feeling—symptoms of a strange phantastic character. Matters which may come up in dreams, in deliria, come up in a relatively clear state of mind. Unreality and absurdity without any evidence of sufficient correctives by sound habits of thought dominate the tantrum. There is a feeling of deficiency or queerness, clearly in contrast with the manic-depressive feeling of mere difficulty and impotence. The emotional reaction is as erratic and poorly controlled as the intellectual appreciation, the ensuing impulses and motor states stamped with a certain monotony, and evidence of cataleptic states.

The attacks usually take the form of a tantrum of some sort, partly characteristic in itself or through the foundation on which it grows. A large proportion stumble over the interpretation of abnormal sensations, or over sexual experiences.

Instead of merely appealing to cortex changes of obscure correlation, or to equally obscure auto-intoxications, or to arrest of development, I refer to the disharmony of habits, disharmony of those regulations which shape a well-balanced economy: The intestinal and circulatory functions, the sexual life, and above all the trend of interests depending in its integrity and efficiency on a certain equilibration. I have been led in my thought by the analogy in the development of morphological phenomena. Roux has shown by his experiments on the mechanics of development how each part of the organism has a certain dynamic and morphogenic possibility, but that in many points the shaping to a final perfection depends on regulation of the balance of the simultaneously growing other organs and their functions. Deficient growth or precocious growth of an organ disturbs these regulations, and the necessary result is a disharmony and every plus is apt to be held up by some minus in another direction. This same principle is eminently valid in functional life, and especially valuable in the most complex of biological regulations—those of mentation. Here a veritably practical and critical presentation of the early work of James has very justly pointed to habits as a unit of observation and biological interest. And it will be our duty to define in actual cases what sets of habits we find interwoven and with what effect. This directs the attention to the investigation of matters which are open to influence in education, and to a more rational

management of dementia præcox, as well as many other mental disorders; and habit-disorder is to be treated by habit-training, not by vague encouragement and excessive protection and mere fighting of incidental disorders. To be sure all incidental disorders, such as the phenomena usually lumped together as auto-intoxication, must be corrected as far as possible, and their correction gives us a vantage ground on which to begin and promote the more fundamental principle—that of habit-training. And in cases where we see disorders developing, whether on ground of heredity or not, it is this issue which guides us in the concrete plan of teaching and prevention. And since the other elements which are apt to figure in our presentations of etiology, nosology and pathology are much more hazy, it is much more satisfactory to come out frankly with a statement that we wish to make distinctions of various types of habit-disorganization, to study the working of the various sets of activities and habits in the patient, determine their relative values by accurate observation coming up to the mark of the experiment, and shaping our therapeutic measures in accord with these principles. This naturally does not exclude in any possible way the consideration of the factors of heredity, and the disorders of this or that organ, but, on the contrary, gives every manageable part its working chance.

In viewing the cases of insanity which are not plainly of exogenous origin, we find certain types of combinations of a more or less distinct symptom-picture, course and outcome. In "An Attempt at Analysis of the Neurotic Constitution," published 1903 as a contribution to the commemorative number of G. Stanley Hall's journal, I specified and briefly characterized the following types: (1) The psychasthenic with its feeling of insufficiency, misplaced tension, apprehension, phobias and impulses, practically as outlined by Janet; (2) the neurasthenic with great fatigability and irritability; (3) the hypochondriacal with feelings of organic insufficiency; (4) the hysterical with dissociations and the characteristic interference by undercurrents of more or less emotionally tinged concepts or thought-habits; (5) the epileptic, fundamentally an abnormal neuromotor reaction type. And finally, a number of groups more closely approaching a certain definite mental derangement: (*a*) The unresistive (responding easily to fever, to intoxication); (*b*) the manic-depressive make-up, described by Hecker;

(*c*) the paranoic type with its tendency to suspicions, interpretations, with or without aggressiveness, and finally, (*d*) the actual deterioration type, which I describe as follows:

In cases of dementia præcox we find over and over an account of frequently exemplary childhood, but a gradual change in the period of emancipation. Close investigation shows, however, often that the exemplary child was exemplary under a rather inadequate ideal, an example of goodness and meekness rather than of strength and determination, with a tendency to keep good in order to avoid frights and struggles. Later religious interest may become very vivid, but also largely in form; a certain disconnection of thought, unaccountable whims make their appearance, and deficient control in matters of ethics and judgment; at home irritability shows itself, often wrapped up in moralizing about the easy-going life of brothers and sisters; sensitiveness to allusions of pleasure, health, etc., drive the patient into seclusion. Headaches, freaky appetite, general malaise, hypochondriacal complaints about the heart, etc., unsteadiness of occupation and inefficiency, day dreaming, and utterly immature philosophizing, and above all, loss of directive energy and initiative without obvious cause, such as well-founded preoccupations, except the inefficient application to actuality. All these traits may be transient, but are usually not mere "neurasthenia," but the beginning of a deterioration, more and more marked by indifference in the emotional life and ambitions, and a peculiar fragmentary type of attention, with all the transitions to the apathetic state of terminal dementia.

I am inclined to put the emphasis on a deficiency of critical and consecutive thought-habits, with a prevalence of interest in the phantastic, mystic, religious and unreal, owing to deficiency in working interests, which would dovetail with the progressive active course of the world.

This scheme is open to many supplements, and it will be an especially grateful task to push the inquiry of individual make-up along the lines of changes of constitutional make-up due to traumatism, to toxic influences, to sexual insufficiency, to the prevalence of certain thought-habits (especially the estrangement with actuality in the form of occultism), and under the influence of the period of involution and senescence.

Clinically it is rather remarkable that the above types keep

fairly distinct. We find in many presentations of neurasthenia, hysteria, etc., the comfortable and probably usually true statement that simple habit-disorganization of most of the above types has nothing to do with insanity, and there need be little fear of its coming on. Experience justifies this comforting remark to a large extent. There is also a certain justification in the strong efforts of many writers to discourage the uncritical intermingling of these types, in terms like hystero-epilepsy, hystero-melancholia. And Kraepelin's effort of taking most of the endogenous deterioration forms into his group of dementia præcox is in part a similar attempt at being systematic. If we take dementia præcox to be a disease by itself which might befall any one, it is not very intelligible; we emphasize in dementia præcox the fact of looseness of judgment and consequential thought with preponderance of habits of the unreal, with either a gradual deterioration of interests, or more acute collapses over difficulties to which the individual is not equal. We might say that the psychasthenic, as a rule, is not of this type, the hysterical is more organized, the true neurasthenic the same, also the manic-depressive type, and in part the paranoic. In the epileptic, deterioration may occur in the form of epileptic deterioration, or as a typical dementia præcox superceding epilepsy. Psychasthenics are apt to deteriorate to the level of lack of initiative of the dementia præcox class. Whether plainly and simply hysterical individuals are apt to develop the characteristics of the dementia præcox complex is denied by some, while Janet claims that every year a few of their cases of hysteria have to be transferred to the service of mental disease on account of deterioration. Many cases of dementia præcox begin with hysterical symptoms. The whole symptomatology of catatonia shows so many traits in common with the phenomena of hysteria and of hypnotism that certain French authors look upon it as a hysteriform psychosis. I should like to refer to a very interesting case that was looked upon as dementia præcox notwithstanding a plainly hysterical onset after confinement, but which recovered completely, and in whom investigation showed that there were none of the manifest and plain antecedents of dementia præcox. This is a patient whom I turned over to a pupil of a prominent worker on hysteria, who declined recognizing in it the hysterical complex and gave a verdict of dementia præcox

without analysis of the facts. I might furnish other instances of tantrums allied to dementia præcox developing without well-marked antecedents of deficiency, and on such ground taking a favorable course.

Looking over the whole field we see in dementia præcox above all the psychic deficiency to meet actuality, a tendency to unreality, to the mystic, common enough outside of dementia præcox, but here combined with the deficiency of judgment and habit due to the undermining effects of other disorganizations of mental and organic habits. The prevalence of defect in the habits of the reproductive zone is most striking, especially in walks of life where the difficulties are less likely to be swallowed up by the muddy stream of open immorality, where conventional morality and frequently excessive observation of superficial morality create remarkable pictures which figure very well as classical representatives of the disorder.

Looking back over the merits of the point of view taken, I should like to say in its favor that it tends towards putting into the center of nosological and pathological attention the only factors which can be of practical importance in the management of these disorders.

In the general discussion on the paragraphs of etiology, these points of disharmonious development are put down as the product of mere fatal constitutional defect, the result of some statistical fate, reminding one of the dogma of infant damnation, training in the students a habit of moving in generalities and fostering a disinclination to going into the study of the actual case; the age at which the disease occurs, the sex, the climate, the stress, and a lot of other things from which no individual can escape are rehearsed, and with a sort of disdain for the actual pathological and morphological value of the directly important things, which are relegated to casual remarks under therapeutics, where we find warnings against masturbation, against overactivity of the artistic imagination, etc.

Didactically and from the point of view of keeping one's self in the frame of soundest activity, it seems to me very desirable that these factors, the working factors of the disease, should be utilized to the utmost; and, in the face of the inevitable criticism that these are old matters which everybody knows and that what

is wanted is some absolutely new discoveries, we need no longer be afraid of conjuring up a moralizing psychiatry if we hold each other down to speaking of the facts as they occur in actual cases, and as they are *not only conceivable but actually at work.* There is hardly anything of which it is not possible to say that the ancient Hindoos, the Greeks and our forefathers thought exactly the same way, and that we do those things in every-day life. Unfortunately, speculation too easily solves many puzzles which it takes many years of experimental and clinical work to put on a safe working basis. It is, nevertheless, concrete work that has to be done and will prove the soundest ground for stimulating the interest of the physician in his work of understanding and modifying cases, and in forming sufficiently definite problems in what otherwise would be a mess of arbitrary creations of nosology.

# CONSTITUTIONAL FACTORS IN THE DEMENTIA PRÆCOX GROUP[1]

By Dr. August Hoch

PSYCHIATRIC INSTITUTE, N. Y. STATE HOSPITALS

Among the present problems of psychiatry the study of dementia præcox is perhaps that which excites the most general interest. It is more particularly the fundamental nature of the disorder which is discussed, and in regard to which the views diverge. The claim is often made that we have in dementia præcox an organic brain disease similar to general paralysis or the other typically organic disorders. There is no doubt that we find in the central nervous system in cases of this group structural changes; some of them are evidently not related to the disorder, while there are others which probably form a part of the process, although we do not yet clearly understand under what conditions they are found nor what their real significance is. While these findings, upon which rests the claim that dementia præcox is an organic disorder in the same sense as is general paralysis, cannot be neglected, and represent a most important field for research, there is another set of data furnished by an analysis of the constitutional factors in these cases, of the development of the symptoms, their nature, and their relationship among each other—data which would seem to show that, granted all the findings of an anatomical and perhaps chemical nature, dementia præcox is after all not a condition which can be placed side by side with the plainly organic diseases, such as general paralysis. Instead of going into a theoretical discussion as to the possibilities of relationship between functional and organic disorders, it would seem wisest, as an introduction to what I have to say regarding the constitutional abnormalities in dementia præcox, to put together the fundamental differences which seem to exist between the plainly organic dis-

[1] Elaborated from a paper read at the New York Psychiatrical Society. November 4, 1908.

orders like general paralysis on the one hand, and the dementia præcox group on the other hand.

In the first place, the dementia in the plainly organic disorders is different from that of dementia præcox. In the former we find that the dementia represents a diffuse disorder of activisation of memories—and hence gives rise to a diffuse defect of elaboration, retention, memory, orientation, etc. In dementia præcox it shows itself essentially in the sphere of interest in the environment, and in a peculiar distortion of the train of thought. In the second place, in the organic disorders we find that the content[2] of the psychosis is of secondary importance, while in dementia præcox the content seems to stand in the very foreground of the clinical picture; the content is, moreover, apt to be peculiarly limited, so that in patients who are accessible to an analysis we find the existence of special trends which account for the entire content. This is something which these cases of dementia præcox have in common with disorders such as hysteria, certain simple paranoic states and some psychoses of degenerates, that is, conditions in which the psychogenic nature of the symptoms is now scarcely questioned by anyone. It is well to remember in this connection that the above mentioned states, to which we shall again have to refer later, are not always easily differentiated from dementia præcox; probably this difficulty exists not merely owing to our inadequate diagnostic facilities, but also owing to the closer relationship which these conditions bear to the disease in question.

Finally, we find that dementia præcox presents yet another side which would point to a certain kinship with these psychogenic disorders. I mean the fact that the relationship between the personality, the special mental make-up of the individual and the psychosis, is a much closer one, the constitutional factors of much more determining importance in the development than in the organic disorders, where we often find either no appreciable peculiarity of mental make-up, or where, at any rate, we have no reason to think that the essential manifestations of the psychosis grow, as it were, out of the personality.

[2] The term content is here used in the sense in which Jung has used it in his "Inhalt der Psychose" (Leipzig u. Wien, Franz Deuticke, 1908), and therefore contrasted to the formal disorders which have received more attention.

It is obvious, therefore, that there are quite a number of important considerations which show that whatever anatomical changes may be found, their relationship to the disorder is evidently not so simple as in those diseases which are clearly organic; and it is therefore necessary to insist that, in addition to a study of anatomical and chemical alterations, there is a vast field of inquiry at least as important and more accessible for the elucidation of the nature of the disorder, namely, the study of the personality and the study of the development and the content of the psychosis.

I wish to dwell in this communication upon the study of the make-up only, and more particularly on the type most frequently seen. It will be remembered that it was Adolf Meyer who, since 1903, has insisted that dementia præcox is a disorder which may not develop in anyone, but that *only some personalities* are in danger, and that in the development inadequate psycho-biological habits play an important part (1).

If we succeed in obtaining accurate anamneses from which we are able to form an opinion of the personality as it existed before the psychosis, or before the incubation period, as it is sometimes called, a certain type of personality recurs with striking frequency, that which I have called the "shut-in personality" (2) the significance of which I wish to discuss more particularly. We may start with a few examples.

One patient, a married woman of nineteen, is said to have been quick enough to learn, and to have studied much; she was also a frequent church-goer, especially since the age of sixteen, without, however, taking an active part in the church work. Even before the age of five she did not get on with other children, did not play with them, but was inclined to keep to herself. When people came to the house she left the room; she was described as always "helpless" in company. She was not liked in school, and never helped others. She was hard to influence—did not take advice. She was over-systematic; wanted things which belonged to her left alone. She was sensitive and cried when interfered with, and was apt not to get over the upset for days. She saw faults in others, rather than her own defects; thought she was better than her fellows, but did not assert herself. She never confided in anyone. She married when eighteen, and during her first pregnancy was uncommonly insistent in her desire to have a boy who should not have red hair, and when it was a girl with red hair she lost interest, and the psychosis at once developed.

The second patient is said, especially since the age of eight, subsequent to an attack of measles, to have grasped her studies less well than

before, was always self-conscious, felt awkward in company and was sensitive, but she said little about it, and in general talked very little. The mother says that the patient was in this respect much like her father, who was, however, a successful, active man. She often sat brooding, was uncommonly systematic and "finicky." At puberty the seclusiveness became more marked. At seventeen she went to a fortune-teller, who told her she would go insane, which prediction is said to have occupied her mind a good deal. At eighteen she fell in love, but her love was not reciprocated. Then she began to grow absent-minded and careless, and gradually drifted into a deterioration.

Case 3 represents a somewhat different type, yet still with well-marked traits pointing in the same direction. The patient, a woman of thirty-six, who always, even as a child, was sensitive and stubborn. She often left the table at the slightest provocation, was hard to guide and influence. It is remembered that she herself would say that she was as immovable as a post. She was married at twenty, did not find her husband congenial, had a tendency to romanticism, never adapted herself to her simple life, made demands upon her husband which she knew he could not meet. Her circumstances as well as her inclinations isolated her. She was moody. Repeatedly she got suspicions of her husband, she fell silently in love with a dentist, had fancies about him, and, for several years before the onset, refused to have any intercourse with her husband. She broke down only at thirty-six, and deteriorated.

These cases suffice to show that we find, in dementia præcox, persons who do not have a natural tendency to be open, and to get into contact with the environment, who are reticent, seclusive, who cannot adapt themselves to situations, who are hard to influence, often sensitive and stubborn, but the latter more in a passive than in an active way. They show little interest in what goes on, often do not participate in the pleasures, cares and pursuits of those about them; although often sensitive they do not let others know what their conflicts are; they do not unburden their minds, are shy, and have a tendency to live in a world of fancies. This is the shut-in personality.

As I have said, this type of make-up is very common in dementia præcox. In a study of my older material from the M'Lean Hospital, Waverly, Mass. (72 cases), I found that in 35 per cent. of the cases it was markedly pronounced, whereas in 16 per cent. it was indicated, so that there was some evidence of it in 51 per cent. When we consider that these findings refer to a time when we did not especially look for these traits, these figures are all the more striking. Other abnormalities of

make-up were noted in 15 per cent.; the make-up was not described in 9 per cent., and in 23 per cent. the claim was made in the anamnesis that the patient's tendencies were "natural." In my more recent material of the Woman's department at Bloomingdale Hospital, N.Y., from which I excluded the cases in which the facts were not accessible—a small material, to be sure, amounting only to 38 cases—I found a typical shut-in personality in 49 per cent. of the cases, indication of it in further 19 per cent., making a total of 68 per cent. altogether. 24 per cent. of the cases are described as showing peculiarities of other types. It is interesting that among the cases who unquestionably deteriorated, the typical shut-in personality was most often seen, occurring in 66 per cent. On the other hand, in the cases who did not show definite deterioration, in other words, who either got well or, while presenting chronic symptoms did not lose their interest in the environment to any marked degree and whose train of thought did not get confused—a group including cases which form a transition to the simpler paranoic states—we found either indications of the shut-in personality or, still more frequently, other abnormalities of make-up, such as long-standing neurasthenoid states, shallowness of emotion, lack of consideration for the environment, or abnormal insistence on precision, a tendency to day-dreaming, evidence of a poorly-balanced sexual instinct; as in one case who for years had drifted into strikingly fruitless love-affairs, another who showed jealousy of the husband for years before the onset and ever since marriage. But I do not propose to go into this part of the topic, as it would lead me too far, and should form the theme of a more extended investigation. Finally, I found only 8 per cent. in which there seemed to be a normal make-up.

Zablocka (3), who studied the dementia præcox material of the Zürich clinic in regard to prognosis, found that the shut-in personality is seen more often in the deteriorating than in the other cases. In this connection it should be remembered that the group of dementia præcox in Zürich is very large, and includes many mild cases which we would not regard as dementia præcox.

Of great interest is an excellent study which Dr. Kirby (4) has recently reported at the New York Academy of Medicine, in which he took up about 100 cases of dementia præcox observed

at the Psychiatric Institute, and found in over 50 per cent. a plainly shut-in personality, and only in a small percentage an apparently normal make-up.

These figures seem to me to tell the story plainly enough. That all cases should show clear indications of a shut-in personality is not to be expected. That as many as 68 per cent. presented evidence of it in my recent material, over 50 per cent. in that of Dr. Kirby, over 50 per cent. in my older cases, and that comparatively few cases show a normal make-up—these are data which cannot be neglected.

The shut-in personality, after all, shows only the direction in which the dangerous traits lie, and it is fair to assume that other abnormalities which we cannot as yet clearly define may work in the same direction. The lack of contact with reality may be only partial, whereas the general response is fair; or other traits which seem to interfere with satisfactory contact, or which foster day-dreaming or which interfere with the formation of objective interests, may be of importance. Kirby has pointed out that a certain shallowness of interest without a general shutting-in was not rarely seen in those of his cases which did not show the typical make-up. Finally, we must not forget that behind a correct appearance, the result of a formal training, there may be much that is not apparent in ordinary life, but which at any time may under stress come to the surface. The fact that these same traits are not seen in manic-depressive insanity is certainly very interesting as a control. How much of it does occur in normal persons is difficult to estimate.

It may not be out of place here to say that in a question of the importance or the danger of certain traits, the whole bearing of which we do not yet know, it is difficult to estimate the limits of elasticity until sufficient strain has been exerted to test it.

We cannot help feeling that to a certain extent these characteristics must in themselves represent a reaction to something more fundamental, but evidently a reaction along a special bent of the personality, and one which owing to its very nature has certain cumulative tendencies.

Freud has shown us that in the neuroses we are dealing primarily with a lack of adaptation to reality in the sexual sphere, with an inadequate or faulty development of the instinct in its

wider meaning, in the sense that owing to a certain fixation and limitation of the interest in that sphere in childhood, and owing to subsequent repression, the later free application and adaptation is interfered with. This is not the place to enter into a discussion of this view which Freud has expressed in his earlier writings, and again very clearly in his recent Worcester lectures (5), and which Jung has extensively supported by his own studies. Important for us in this discussion is the fact that Jung has insisted that a similar sort of abnormality in the sphere of the sexual instinct exists in dementia præcox. While I have personally not analyzed a sufficient material with the view of verifying this claim from direct evidence, I am nevertheless inclined to regard this as very probably a correct one, for the following reasons: In the first place, we should mention the close relationship which exists between dementia præcox and puberty. Secondly, the fact that everyone who has attempted to enter into the lives and struggles of these patients must have been impressed with the frequency with which sexual conflicts are found to have played an important rôle in the development of the disorder. Thirdly, analysis of the content of the psychosis shows us again and again the existence of sexual trends, and often when the sexuality manifests itself, it does so in a peculiarly diffuse, poorly adapted manner, such as in the falling in love with several persons at the same time, and the like. All this cannot be accidental. There is, therefore, much that speaks in favor of the claim of a fundamental lack of sexual adaptability; but, according to those who have analysed the neuroses of various kinds, we find there similar difficulties, but without equally serious consequences.

We are scarcely prepared to say what is fundamentally the nature of the lack of sexual adaptability, and what is its relation to the shut-in personality. However, Abraham (6), who has fully recognized the importance of the shut-in personality, has attempted to answer this question. He regards the lack of sexual adaptability as due to an arrest of sexual development, a permanent retention of the infantile autoerotic stage, and looks upon the shut-in tendency as one of the expressions of this autoerotism. This is certainly a clever hypothesis, but we cannot help feeling that the question is more complicated.

But even without knowing what is the origin or the funda-

mental meaning of the constitution we have described, we can see, nevertheless, reasons why such a constitution should represent a serious menace to the mental balance of the personality.

The inability to get into contact with the environment bears in itself many dangers; it prevents an active aggressive shaping of the situation which is so important for the progress of the normal individual and which forestalls further conflicts; it prevents the corrective influences which actual experience constantly furnishes, and which is gained in the mingling with people, the mutual actions and reactions; it fosters the growth of unproductive fancies. Everyone has a certain inclination to day-dreaming, but, aside from the fact that it plays a rather subsidiary rôle in the normal robust person, the fancies often represent in them the first dim outlines of future plans, and therefore are not without reference to reality, and receive their value from that side; but fancies which are out of contact with reality probably exert in themselves a certain fascination which progressively limits objective interest. Moreover, these very tendencies make the individual unfit to acquire those constructive plans and hopes, not necessarily elaborated, but felt, dimly appreciated, upon which the normal person lives, and which give to him the very essence of his existence. There is an absence of that progressive, prospective satisfaction which cannot be too much insisted upon as necessary for the retention of mental health. The active contact with the world makes, of course, more demands upon the individual than a life in pure fancies, towards which the path of least resistance evidently leads in these patients. We see, therefore, that the traits upon which we would lay most stress in the shut-in personality, the lack of contact with the environment, the satisfaction with fancies instead of objective interests, the lack of constructive aims and aggressiveness, must have dangers in them which it would be difficult to exclude as dynamic factors in the development of these disorders, and we must agree with Adolf Meyer when he has again and again insisted upon the importance of faulty psycho-biological habits in connection with dementia præcox.

It will now be of interest to briefly review some constitutional abnormalities in groups of cases which present a certain kinship to dementia præcox,—I mean in hysteria, certain paranoic states,

and the psychoses of degenerates recently reported by Birnbaum; cases therefore in which, and this is the point I wish to emphasize more particularly, deterioration does not occur. In hysteria, in which disorder we have, as was above stated, according to Freud, also a certain lack of sexual adaptability, we find a very different sort of personality. Here there is no lack of aggressiveness, nor lack of contact with the environment, nor an absence of objective interest; on the contrary, we find a constant reference to the persons about, a desire to be in the centre of observation; hysterical patients force us with all means at their disposal to occupy ourselves with them.

In paranoic states, too, the contact with the environment is plain; these persons are sensitive, and markedly concerned about the rest of the world, they expect something from it, and with all their suspiciousness they are not without a certain open attitude in the sense of aggressiveness and a desire to seek contact. Another equally important difference between the paranoic state and the condition of dementia præcox is to be found in the fact that sometimes the external situation is a much more potent factor in the causal constellation of the former, as is seen in the paranoia quærulans, and other paranoic states, such as those reported by Gierlich (7) and Friedmann (8), or those more recently by Rüdin (9).

We may finally consider that interesting group of cases which lately has been taken up by Birnbaum (10), the first German who fully recognized the claims of the French school regarding the *délire des dégénérés*. It is interesting that these are psychoses which often resemble in their mechanisms those of dementia præcox, so much so that Bleuler (11), one of the investigators best acquainted with the psychology of dementia præcox, has met Birnbaum with the statement that the latter had not succeeded in giving a differentiation beween his cases and dementia præcox. To this Birnbaum has given, what I consider a satisfactory reply:[3] there is much that separates the two groups if one considers well-marked cases of either group, but it does not seem unlikely that transitions occur, although plainer transitions exist between these psychoses of degenerates and hysteria.

[3] In Centralblatt f. Nervenheilkunde und Psychiatrie, 1909, p. 429, although I cannot by any means agree with his entire position.

In the group which Birnbaum has described we find individuals with criminal tendencies who, under the influence of punishment, imprisonment, frustrated escapes, denials of pardon, announcement of a new indictment, etc., developed psychoses with delusions, hallucinations, and other symptoms in which the element of wish-fulfilment takes a very prominent and easily recognized place. The disorder, then, depends strikingly upon external difficulties and often disappears with the removal of the latter. As to the make-up, these personalities, besides presenting criminal tendencies, are unstable, without much determination or depth of feeling, though with a tendency to outbursts of feelings; they are fickle, frivolous, unable to stick to any occupation, suggestible, imaginative, eccentric, given to fantastic schemes, untruthful. These patients, then, do not show a lack of contact with the environment, they even present a certain ill-directed aggressiveness. They are, therefore, in many ways different from individuals with a shut-in personality. In another point they differ, namely, in the fact that their conflicts are on the surface; they are in a scrape, to put it tersely, and the content of the psychosis is a reaction to the scrape. Hence the make-up as well as the situation differs from that of dementia præcox, although the mechanisms are, as we said, often not unlike those seen in dementia præcox.

This putting side by side of the constitutional traits of hysteria, paranoic states, and certain psychoses of degenerates on the one, and dementia præcox on the other hand; this contrast of groups of cases which show no tendency to deterioration with a group of cases in which deterioration is an important feature, seemed to me to be of value, because it can hardly be regarded as accidental that those who deteriorate show original defects in the direction which we have indicated, while those who do not deteriorate are singularly free from those very traits; nor is the prominence of external factors in paranoic states and in the degenerates, in contradistinction to the essentially internal conflicts in dementia præcox, likely to be accidental and without bearing on the question of the outcome.

Having then shown the prevalence of the shut-in personality in dementia præcox, and its absence in the non-deteriorating cases,

it is left for us to point out certain relationships between the traits we have brought out and the symptom pictures early and late.

In the first place, it is well known that the incubation period, if we may be allowed the term—a period that often lasts several years—is almost always marked by an accentuation of the shut-in traits, the patient gets further away from the environment. Secondly, we can scarcely help seeing the close relationship which exists between the constitutional traits and the negativism, and last, but not least, between these traits and the final deterioration. What is, after all, the deterioration in dementia præcox if not the expression of the constitutional tendencies in their extreme form, a shutting-out of the outside world, a deterioration of interests in the environment, a living in a world apart?

All these considerations will, I hope, make clear that the constitutional abnormalities which we have described, and which in their most marked form probably represent the direction in which the important traits lie, must be the expression of dynamic forces of great importance.

## References

1. Adolf Meyer. An Attempt at Analysis of the Neurotic Constitution, Amer. Journ. of Psychol., Vol. XIV, pp. 90–103, 1903. Also Fundamental Conceptions of Dementia præcox, British Medical Journal, Sept. 20, 1906; The Dynamic Interpretation of Dementia Præcox, Amer. Journ. of Psychol., July 1910, Vol. XXI, pp. 385–403.

2. Hoch. A Study of the Mental Make-up in the Functional Psychoses. Abstract of paper read before the New York Psychiatrical Society, Nov. 4, 1908. Journ. of Nervous and Mental Disease, April, 1909, Vol. XXXVI, p. 230.

3. Zablocka. Zur Prognosestellung der Dementia Præcox, Allgemeine Zeitschr. f. Psychiatrie, Vol. LXXV, pp. 318–339.

4. Not published.

5. Freud. Drei Abhandlungen zur Sexualtheorie, and Über Psychoanalyse. Franz Deuticke, Wien, 1910. This Monograph Series. No. 7 in Translation.

6. Abraham. Die psychosexuellen Differenzen der Hysterie und der Dementia Præcox, Centralblatt f. Nervenheilkunde und Psychiatrie, July, 1908.

7. Gierlich. Über periodische Paranoia und die Entstehung der paranoiden Wahnideen, Arch. f. Psychiatrie, Vol. I, pp. 19–40, 1905.

8. Friedmann. Beiträge zur Lehre von der Paranoia, Monatsschr. f. Psychiatrie und Neurologie, Vol. XVII, pp. 468, 532.

These two translated in this Monograph Series No. 2.

9. Rüdin. Über die klinischen Formen der Seelenstörungen bei zu lebenslänglicher Zuchthausstrafe Verurteilten. München, 1909. Review Centralblatt für Nervenheilkunde und Psychiatrie, 1909, p. 292.

10. Birnbaum. Psychosen mit Wahnbildung und wahnhafte Einbildungen bei Degenerativen. Carl Marhold, Halle, 1908.

11. Bleuler. Wahnhafte Einbildungen der Degenerierten, Centralblatt f. Psychiatrie u. Neurologie, 1909, pp. 77–80.

# COMPARATIVE PSYCHOLOGICAL STUDIES OF THE MENTAL CAPACITY IN CASES OF DEMENTIA PRÆCOX AND ALCOHOLIC INSANITY[1]

By Henry A. Cotton, M.D.,

MEDICAL DIRECTOR, NEW JERSEY STATE HOSPITAL AT TRENTON

(From the Psychological Laboratory of the Royal Psychiatric Clinic at Munich. Professor Kraepelin, Director)

## Scope and Object of the Investigation

The work of the investigators in the field of experimental psychology in recent years has shown remarkable progress, so that new light has been shed upon a great many obscure and complicated problems connected with our mental life.

Mere speculation as to the mechanism of fundamental psychical processes has given place to some extent to more accurate knowledge of these problems, so that today many obscure and complicated reactions can be graphically demonstrated and these same processes spoken of in terms of mechanical values and equivalents. To reduce these complicated processes to figures and curves has been a stupendous task in the field of normal psychology, and much remains to be accomplished before we can have an accurate idea of all psychical phenomena.

That the experiments with normal individuals have not been entirely successful is due partly to a lack of methods, and partly to defects in some of the methods devised. If this be true of the field of normal psychology, then we cannot wonder that investigators in the field of abnormal psychology have experienced great difficulties in representing by graphic methods the complicated abnormal psychical processes in the insane.

The effects of drugs and poisons upon the psychical processes have been graphically shown by Kraepelin and his pupils, and their work has been of immense importance in paving the way

[1] Read before the New York Psychiatrical Society.

for a better understanding of abnormal psychological phenomena. But experiments with normal individuals do not present such obstacles as we find when we attempt to analyze abnormal psychical processes of the insane by similar methods. The normal processes with and without the effect of the poisons can be carefully analyzed and compared in the same individual, whereas, in the experiments in abnormal psychology, we must compare similar experiments upon normal individuals with the results in abnormal individuals or the insane.

Methods that are readily adapted to the normal individual become useless when applied to investigations of abnormal mental conditions as found in the insane. Either the patients absolutely refuse to perform the experiments, or only partially fulfill the conditions, and the figures and results of such experiments are not comparable with similar experiments with the normal. And such figures seldom give an accurate and clear picture of the pathological mental condition.

We have studied the various clinical symptoms of the insane, and we know that certain symptoms refer to disturbances of certain psychical fields. The problem of experimental psychology is to measure these abnormal reactions and compare the results of such measurements with those obtained in normal psychical processes.

For many years Kraepelin has searched for simple methods which were suitable for abnormal as well as normal persons, and he and his pupils have been successful in finding methods that on the one hand were suitable for studying the psychical processes of normal persons, while on the other hand, they were adaptable to abnormal psychical processes as well.

His method of continuous addition of single numbers was first used in experiments with normal individuals, and occupied two hours a day continuously for its completion. By this simple method he succeeded in graphically representing some very important psychical processes, and also the relation of various processes to each other, i. e., fatigue—the effect of the rest or pause during addition, in which recuperation takes place; the effect of stimulation; the impulse and intensity of the will, and variations in the will; and the increase of actual work done through practice and familiarity with the work. Kraepelin has

shown this in his monograph on the "Arbeitscurve,"[2] and in one chart all these various processes are plotted, and can be graphically compared. Kraepelin admits that it took ten years of experimenting with normal individuals before he could interpret all the facts shown by the "Arbeitscurve." Gradually Kraepelin shortened the time from two hours to one hour, then half an hour, until finally the experiment was reduced to ten minutes. It was soon apparent that two hours continuous addition was very fatiguing to a normal individual and wholly unsuited to abnormal ones.

Through a series of experiments extending over these ten years it was found that the length of time in the various experiments had little to do with the relation of the various phenomena; that certain laws were uniform, whether the experiment was for two hours or ten minutes, and that the relation of these phenomena were shown to be constant.

The method used in investigations by the author of this paper is practically the original method reduced to ten minutes work and simplified. And in spite of the simplicity and crudeness of the method, it has been used with success to show at least some of the abnormal psychical processes of the insane.

The object of this work was to investigate two forms of psychoses by this method, i. e., dementia præcox and alcoholic psychoses, and to compare the results obtained, with the results of the experiments on normal individuals of the same station in life and grade of intelligence. The method is simply a continuous addition of single numbers. These numbers are placed in long columns, and the task of the patient is to add consecutive numbers together and place the result opposite these numbers. This continues for ten minutes, and at the end of each minute, by a signal, the patient makes a line to indicate the same. The experiment lasts ten days. On alternate days a five minutes' pause or rest is given, and on other days the patient adds for ten minutes without any pause or rest. Then the number of additions during each minute is taken as units of the curve. Experience has shown that the amount of time lost in writing the result is very small, and extends uniformly over the entire experiment, and need not be

[2] Die Arbeitscurve, Emil Kraepelin, Leipzig, 1902.

taken into account, also, that mistakes made need not be considered, as the task is so simple that any one who has a rudimentary education in arithmetic can undertake the experiment.

This investigation was begun in the Psychological Laboratory of the Royal Psychiatric Clinic in Munich in the spring of 1906. The patients for investigation were partly from the Psychiatric Clinic in Munich, and partly from the District Insane Hospital at Egelfing, in the vicinity of Munich. The experiment with the patients is a comparatively simple matter, but calculating the results and interpreting the same is extremely difficult, and to Professor Kraepelin should be given the credit for a large share of the work, for without his assistance in interpreting the results the experiment would have been worthless.

We have investigated altogether fourteen cases of alcoholic psychoses, among which were several cases of delirium tremens, alcoholic hallucinosis, and chronic alcoholic insanity. Also twelve patients with dementia præcox who were in the first stage of the disease, mostly the catatonic form. The figures for the *normal* were taken from the work of Drs. Plaut and Rehm of the Psychiatric Clinic in Munich, who investigated these normal cases in conjunction with similar work with manic depressive insanity and psychasthenia.

I am indebted to these men for furnishing me with figures of normal people, and for their assistance in preparing this work. I wish also to express my thanks for the courtesy of the assistants in the Clinic of Munich, as well as the assistants in the District Insane Hospital at Egelfing, who rendered much aid in providing suitable patients for these studies.

## The Method in Detail

As we have mentioned before, the method consists in adding single figures together and placing the sum opposite the printed numbers. Thus, a page of printed figures is before the patient, arranged in long columns, about 50 figures to a column.

These experiments are performed every day for 10 days. On alternate days a rest of five minutes is allowed, so that the rest is indicated by a dotted line between the fifth and sixth minute (see Fig. 1).

Thus—

| | |
|---|---|
| 8 | |
| 9 | 17 |
| 3 | 12 |
| 4 | 7 |
| 6 | 10 |
| 2 | 8 |
| 5 | 7 |

the left hand figures being the printed figures, and the right hand figures the additions of consecutive numbers. A stop watch is held by the investigator, and at the end of every minute the patient is told to "mark," which he does, and goes on adding until the 10 minutes are finished.

The curves shown in Fig. 1, are made up as follows: The abscissa represent the minutes of work from 1 to 10, and the ordinate represents the total number of additions performed in each minute. The curve on the left "without pause," is made from the work on alternate days, first, third, fifth, etc., days, and the curve on the right "with pause," is made up from the work on the second, fourth, sixth, etc., days. These curves represent the average of each five days' work. To illustrate, one example of the work done on normal persons by Wilhelm Specht. Below are given the numbers for each minute in the ten days' investigation.

| 1. | | 3. | | 5. | | 7. | | 9. | |
|---|---|---|---|---|---|---|---|---|---|
| 63 | 54 | 75 | 66 | 73 | 69 | 75 | 73 | 78 | 75 |
| 56 | 52 | 69 | 67 | 71 | 66 | 70 | 71 | 73 | 71 |
| 58 | 56 | 64 | 68 | 69 | 67 | 70 | 72 | 75 | 66 |
| 53 | 52 | 68 | 66 | 70 | 68 | 72 | 74 | 73 | 66 |
| 54 | 52 | 68 | 62 | 73 | 68 | 73 | 70 | 71 | 65 |
| 284 | 266 | 344 | 329 | 356 | 338 | 360 | 360 | 370 | 343 |

| 2 | | 4 | | 6 | | 8 | | 10 | |
|---|---|---|---|---|---|---|---|---|---|
| 48 | 61 | 72 | 69 | 73 | 76 | 77 | 80 | 74 | 76 |
| 44 | 55 | 64 | 66 | 67 | 72 | 74 | 72 | 74 | 75 |
| 43 | 51 | 57 | 66 | 70 | 70 | 74 | 67 | 75 | 74 |
| 44 | 47 | 63 | 64 | 67 | 69 | 70 | 70 | 73 | 72 |
| 44 | 50 | 59 | 64 | 68 | 69 | 70 | 70 | 72 | 74 |
| 223 | 264 | 315 | 329 | 345 | 356 | 365 | 359 | 368 | 371 |

In the above table the vertical figures represent the number of additions in minutes, the first five minutes on the left of the vertical line, and the second five minutes on the right, and the successive days, as explained above. The average of these daily figures is shown in the two curves—in Fig. 1 we see in curve *b* (with pause) that there is a decided downward course of the curve, showing a drop from 42.5 to 38.7 at the end of the fifth minute. The cause of this sinking of the curve is due principally

to fatigue (mental) as the mental work progresses. But there is another factor to be considered, and that is practice. For practice without fatigue would show an increase in the amount of work done, and the curve would rise instead of falling. But the fatigue overbalances the practice and consequently the curve sinks.

After a five minutes pause, it will be noticed that the work

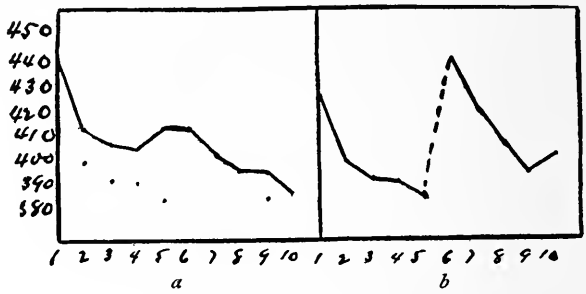


FIG. 1.

done in the sixth minute is not only much greater in amount than at the fifth minute (directly before the pause), but is also larger than the amount done at the beginning of the experiment, and consequently we find the curve beginning almost at 44 instead of 42 the first minute.

This increase in the amount of work done the sixth minute is explained by two facts which have taken place during the five minutes' rest or pause. In the first place the fatigue has been overcome by recuperation. and the person starts out freshened and ready to work. At the same time the effect of the practice of the first five minutes has some value in the amount of work done, being more than in the first minute. It is true that during the pause or rest, the practice has to a small degree lost its effect, but the disappearance of fatigue also must be considered in explaining the larger value of the work in the sixth minute. Especially is this seen in comparing the work of the sixth minute in curve *a*. Here the value is 41 against 44 for the same minute after a pause of five minutes. The work of the entire five minutes after the rest is much greater than the work of the first five minutes. Hence, we must ascribe the increase largely to the effect of recuperation during the rest, and the loss of fatigue.

From Table I we get the following values: The numbers on the left side of the line are the values of the fifth minute before the pause for five days. On the right side of the line are the values for the sixth minute following the pause for five days. The sum of these separate columns gives the total amount of work done in the fifth and sixth minute, with an intervening pause.

| | |
|---|---|
| 44 | 61 |
| 59 | 69 |
| 68 | 76 |
| 70 | 80 |
| 72 | 76 |
| 313 | 362 = 11.5 per cent. increase after the rest. |

Here is shown distinctly the effect of the pause or rest in the 11.5 per cent. increase in work done in the sixth minute over that done in the fifth minute before the pause. When we compare the work done in the fifth and sixth minutes on the days with the pause with the same minutes on the days without the pause, we see in curve *a* that the fifth and sixth minutes are practically the same, that no increase can be noticed as compared with curve *b*. For the fatigue has been compensated in the latter, but not in the former instance. The curve shows in general a descending tendency due, as we stated above, to the overbalancing of the fatigue over the practice, but the fifth and sixth minutes are at the same level. The slight rise of the curve at the fifth minute is not due to a balance between the fatigue and practice, but to the effect of the tension, or straining of the will, which overcomes the fatigue. And the straining of the will frequently shows itself in the curves and is considered an impulse of the will to overcome the fatigue. This impulse is effective in keeping the amount done in the fifth and sixth minute at the same level. Without this straining of the will in the course of the work, both values of the fifth and sixth minutes would be much smaller and the curve would descend at this point as a result of fatigue. So that 11.5 per cent. increase after the pause stands for the direct effect of the pause, when taken with the percentage of increase on the days without the pause, thus:

11.5 per cent. increase after pause.
0.0 per cent. increase without pause.
11.5 per cent. increase after pause, or direct effect of pause.

We must also consider the total amount of work done before the pause and after the pause or the total work of the first five minutes must be compared with the total work of the five minutes after the pause, and also compare these percentages with those obtained in the same manner by comparing the same values of the days without a pause.

From the table we get the following figures, the left hand column representing the total work of the first five minutes for five days, and on the right side, the total work of the last five minutes after the pause.

| | |
|---|---|
| 223 | 264 |
| 315 | 329 |
| 345 | 356 |
| 365 | 359 |
| 368 | 371 |
| 1,616 | 1,679 = 3.3 per cent. increase of total work after the pause. |

This increase after the pause is explained by the effect of the practice, a portion of which remains even during the rest, and therefore more work is accomplished in the last five minutes, although the fatigue also plays a part as shown by the descending curve. When we assume that during the pause the fatigue has been entirely recuperated by the rest, then the increase in work after the pause represents the practice coefficient of the individual.

Under what conditions, and in what manner the fatigue is compensated during the pause, at present we are unable to state.

Through the increase of the work after the pause, although a certain residual of fatigue is present, we must conclude that the practice has overcome to some extent the fatigue. The residual can be great, and at the same time hidden by the effect of practice when the latter is sufficiently great. And we can also conclude that possibly during the rest or pause, that the fatigue is entirely compensated, and no residual remains, and that the effect of practice is very small.

To come to a definite conclusion in regard to this question, we must first compare the work equivalents of the days when no rest was taken, and where consequently the effect of the rest does not come into play. From the table we again take the following

figures. The figures on the left represent the total work of the first five minutes for five days, and on the right of the column, the total work for the last five minutes for five days.

| | |
|---|---|
| 284 | 366 |
| 344 | 329 |
| 356 | 338 |
| 360 | 300 |
| 370 | 343 |
| 1,714 | 1,636 = 4.1 per cent. decrease in last five minutes over first five minutes. |

In other words, without the good effect of the rest, the fatigue shows itself, and the work fell off 4.1 per cent. But from this coefficient alone we cannot compute the effect of fatigue. In the second five minutes work the fatigue overcomes everything else, but we cannot tell to what extent the total amount of work has been influenced by the simultaneous opposing effect of the practice.

It is possible that the decrease of the work in the second five minutes might be much less, if the individual was capable of improving by practice. We get an idea of the importance of the fatigue when we compare the differences in the total amount of work done after the pause and without the pause.

That the work of the second five minutes on the days without and with a pause or rest, approach each other as regards the effect of practice, can be shown. However, where recuperation of the fatigue during the pause has taken place, the comparison of days without any rest (where the second five minutes is under the influence of the fatigue) and the days with rest, the difference in the two series will give us the value of the fatigue. This difference is shown below.

3.3 per cent. increase in total work after pause.
4.1 per cent. increase in total work last five minutes.
7.4 per cent. coefficient of fatigue.

For computing the individual fatigue we make use of two groups of figures obtained by this method from the same individual. On one hand the relation of work values of the fifth and sixth minutes, and on the other hand, the difference in the total

amount of work done in the first and second five minutes on the days with and without pause. We will see later, when discussing fatigue in our three groups, how the coefficient of fatigue is vastly different in dementia præcox from normal individuals and alcoholics.

### Comparison of Individual Work Curves

In Fig. 2 we have shown a typical curve from the groups investigated and compared with a normal curve.[3]

The curves on the left each represent the average of five days work for ten minutes per day uninterrupted. The curves on the right represent the average of five days work with a pause of five minutes after the fifth minute of work. This period is represented by the dotted line. The curve of the following five minutes represents work after the pause.

In the normal curve *a–I* it will be seen that there is a gradual tendency of the curve to descend, although in places it appears almost horizontal. However, the values do show that there is a decrease in amount of work done in successive minutes, but in tenths so that it cannot be accurately shown in the curve. There is a certain regularity to the curve, when compared to that of dementia præcox. Also in curve *a–II* (normal), the effect of the pause is distinctly shown, for after the pause, presented by the dotted line, the curve begins at a much higher point than the fifth minute, and a trifle higher than the beginning of the curve. This curve also shows the gradual decline in the amount of work done, both before the pause and after the pause, although the entire curve after the pause is distinctly higher than the curve of the first five minutes. As we said above, this is due to the loss of fatigue during the rest, and also to the residual practice, which has shown itself in the period following the rest.

Comparing this curve of a normal individual with curve *b,* that of a dementia præcox case, the difference between the two is at once apparent. In the first place one sees tremendous variations in the curve *b–I*. The curve sinks very low at the fifth minute,

[3] Curve taken from figures of a normal person given by Wilhelm Specht in Über klinische Ermüdungsmessungen, Archiv für die Gesamte Psychologie, Band III, Heft 3.

then rises again and ends much higher. This can only be accounted for by the variations in the intensity and impulse of the will, of which we will speak more in detail later. When we observe the curve *b–II,* representing the period with a rest, we

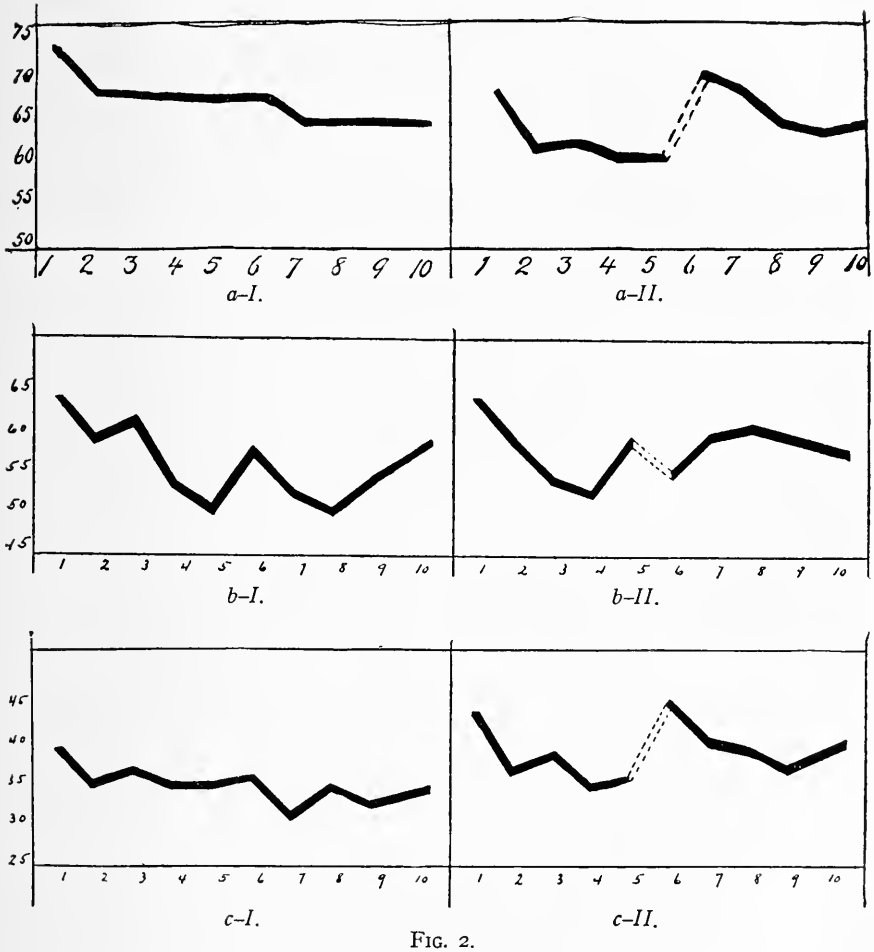


FIG. 2.

see that instead of the sixth minute rising distinctly above the fifth minute (before pause) the curve starts at a much lower level. This shows that the rest has not had a good effect upon the work of the patient, but has had a decidedly unfavorable

influence on the after work. It also shows that there was either no fatigue present or that if present, it was not compensated during the pause. And from what we know clinically of dementia præcox cases, especially the catatonic forms, the first supposition is the correct one. The sinking of the curve, then, is not due to fatigue, but to fluctuation and variations in the intensity of the will; although apparently working steadily along, the curve shows that the will is far from being under the control of the patient.

In curve *C–I* and *II,* that of a patient with alcoholic hallucinosis, we find very little deviation from the normal. The fatigue is shown by the gradual descent of the curve. And after the pause or rest, the curve begins at a much higher level, not only as compared with the fifth minute, but with the first minute as well. While these curves are shown as typical of the three groups under discussion, it must not be supposed that all of the cases in the three groups would show similar curves respectively in each group. There are wide variations in individuals of the three groups, but at the same time the average of the curves of each of the three groups will show distinct differences from each separate group. Especially will this be shown later when different factors are considered, such as fatigue, impulse of the will, daily increase in work, total amount of work done, etc.

### Combined Average Work Curves (Fig. 3)

An effort was made to show the average curve of each group, but the results of averaging all the curves of one group was not a representative curve, as the variations of each individual curve would balance that of the others of the same group, and the result was not a representative curve. But this difficulty was overcome by Professor Kraepelin, when he suggested curves made up of the relation of each minute to the first minute, without any reference to actual values. So the combined work curves were constructed as shown in Fig. 3. Here the peculiarities of each group were represented graphically, and the result was startling. The work of the first minute in each group is the basis for computing the proportion of every other minute to the first. The standard value of the first minute is placed at 100. The value of the other minutes are computed by logarithms in terms of the first

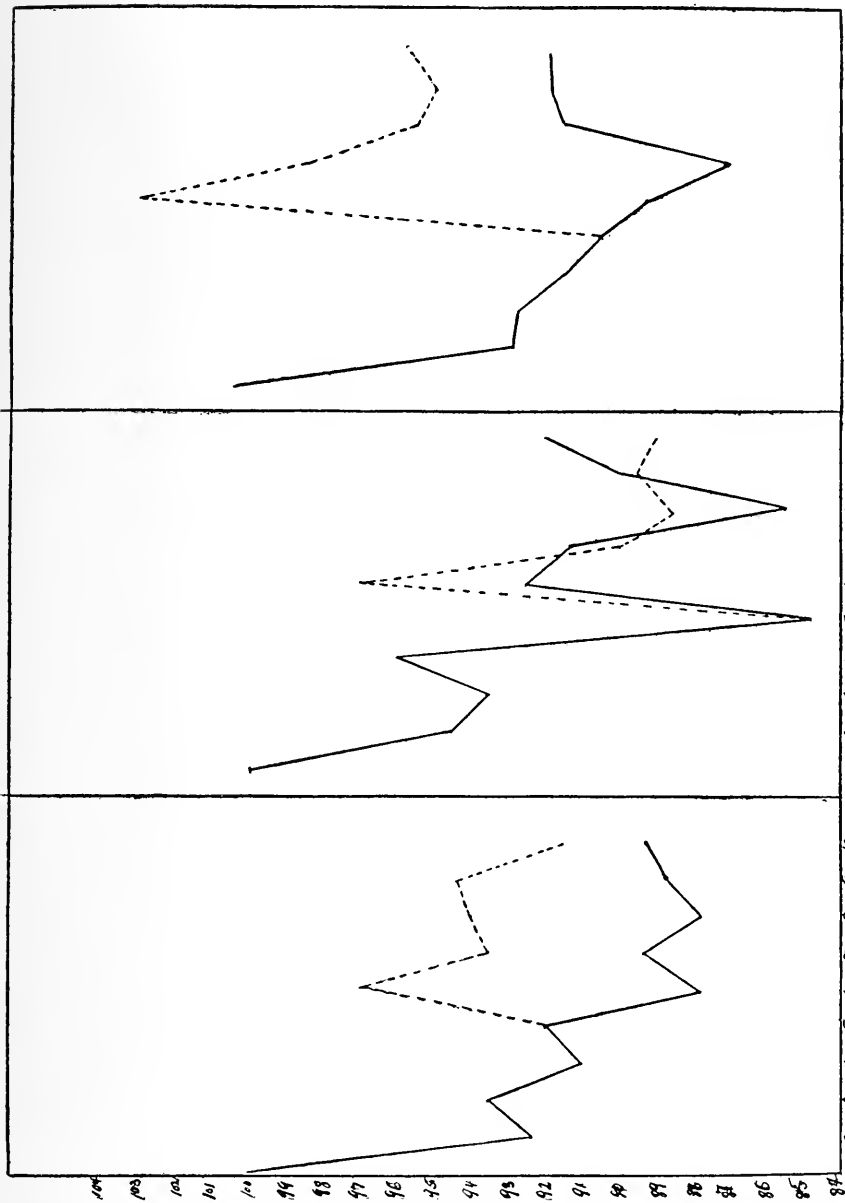


FIG. 3. Combined average work curves of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

minute, or 100. Thus, in the normal curve, constructed from the average proportion of the 25 individuals, shows the second minute to be 92.4, in other words, the relation of the average of the first minute to the relation of the average of the second minute of all individuals, is as 100 to 92.4, and so on for the rest of the curve. And the same method was used for the other two groups. It will be noticed that at the fifth minute the curve divides, one curve continues on, and the other is represented by a dotted line. This division of the curve is to show the difference in the relations of the last five minutes without a pause (the uninterrupted line), and the last five minutes following the pause or rest, as shown by the dotted line. The first five minutes of each day is taken, as the work is uniform until the end of the fifth minute, when on one day a pause intervenes, and on alternate days no pause is allowed. In other words, the first five minutes of all curves of an individual group are obtained under similar conditions, while after the fifth minute, the pause makes a distinction. And the effect of the pause is graphically shown by the curve represented by the dotted line. In the normal it will be seen that the curve gradually descends, although there are some slight variations throughout the ten minutes work without a pause. (Note the abscissa indicates the minute of work, the ordinate the proportions of each minute to the first minute.) The sixth minute (dotted line) is separated from the sixth minute (straight line) by a considerable space. In the former the value is 97, in the latter 88. This difference represents the direct effect of the pause, and it will be noted that the course of the curve (dotted line) continues at a much higher level than the other curve. This corresponds with the typical normal curve in Fig. 1. Another peculiarity is that in the last two minutes of the lower curve there is a slight rise, showing the effect of the straining of the will by a distinct effect to overcome the fatigue. As the fatigue after the pause is less, we do not have this strain at the last, and the curve sinks during the last minute of work.

The difference between the values of the first and second minute represents the impulse of the will (Antrieb). As we all know, when one has a certain task to perform one goes at it with a strong impulse to accomplish the task, and this tension, as soon as the work is started, lets up, and the second minute shows a

considerable decrease in the amount of work done as compared with the first minute, or at the beginning of the task. And the difference between the sixth and seventh minute after the pause (dotted line) represents the impulse of the will after the pause. This is not so great as the impulse at the beginning, as through practice the task is known, and one does not strain the will to accomplish the work at hand to such an extent as first.

When we observe the combined curve of the dementia præcox cases, we at once notice a marked contrast with the normal. The same variations and fluctuations in the curve are seen as were seen in the typical curve of dementia præcox in Fig. 1, *b*. But this is a composite curve of the twelve cases of dementia præcox, and the variations would be lessened rather than increased. One point of difference between this and the normal curve is the sudden sinking of the curve from the fourth to the fifth minute. Here is a drop from 96.5 to 84 and then a sudden rise to 93 at the sixth minute. As seen by the fluctuations, the question of fatigue does not come into consideration at all. First, because there are two ascents to the curve following this, and secondly, because the sixth minute after pause is comparatively very little above the sixth minute without any pause.

To find the explanation, then, we must consider the will which is seriously affected in dementia præcox. Here the defect is graphically demonstrated, and the sudden descent of the curve can be ascribed to the neglect of the will (Vernachlässigung des Willens) or deflection of the will.

The fluctuations of the curve in the last five minutes (without pause) is remarkable and is explained by the difficulty of keeping dementia præcox cases at a given task. The apathy shown clinically is here graphically demonstrated.

The course of the curve after the pause (dotted lines) also bears out the statement that there is little fatigue, and the curve, instead of remaining at a higher level after the pause, actually sinks lower than the continuous curve. So that from this curve three facts are demonstrated: (1) The absence of fatigue, (2) the irregularity of the tension of the will, (3) the absolute deflection of the will. The impulse of the will at the onset of the work is much less than in the normal and much less than that in alcoholics. The third curve represents the composite curve of the alcoholics.

This type of patients investigated were variable, and some were practically normal, especially those recovering from delirium tremens. The others belonged to the class of alcoholic hallucinosis and alcoholic paranoic conditions.

This curve does not differ materially from the normal, except in the height of the curve at the sixth minute after the pause. Here the curve starts at 103 or 3 points above the height at the beginning of the work and six points above the same value in the normal curve.

This increase in the amount of work done in the sixth minute in alcoholics over the normal is explained, not so much by the fatigue, but by the fact that during the pause the residual idea of the work is lost, and they start in with a marked tension of the will, assisted by the residual practice-effect (Übungs Nachwirkung). And they seem to have lost the impression of the work during the pause, so that it is in one sense a new task to them, while in the normal the memory impression is more lasting, and the normal does not start out with such a great impulse to accomplish the task.

## Total Mental Capacity

### (*Absolute Leistung*)

We obtain the values for the total capacity for mental work or the absolute ability for work in the various groups, by taking an average of the total number of additions performed in the first five minutes for the ten days. It is impossible to take the average of the whole ten minutes for on alternate days a rest or pause is allowed after the fifth minute, and consequently the last five minutes of work cannot be used in computing the amount of work performed by the individuals of the various groups.

By observing Chart I we will see the values for the total mental capacity represented in a graphic manner. The values are represented by columns for comparison, and it will be seen that there is very little difference in the amount of work done by the three groups. The values for the normal group vary from 263 to 104, and the average for the group is 185. In the dementia præcox group the variations are between wider limits, i. e., from 289 to 92, but the average is considerably lower than in the case of the normal group, viz., 165.

In the alcoholic group the variations are between much wider limits, i. e., from 323 to 96, and the average for the group is 188, a trifle higher than the normal average, while the average for the dementia præcox group is much below that of the normal and alcoholic groups. One is at first surprised that the difference is so slight. But when we consider the clinical features of dementia præcox, this finding is not so surprising, and the clinical and psychological facts are in harmony. We know that frequently in this class of cases the intellectual defects are slight, and often absent for many years, so that the patients afflicted with this disease retain to a considerable degree their intellectual faculties, although profound disturbance of other psychic fields (emotion, will and ideation) are present and prevent the patient from living a normal mental life.

We will demonstrate later that the disturbance in dementia præcox is to be found in other fields than the purely intellectual. Of course, in the end stages of the disease this field also suffers and we see cases with profound mental deterioration. The alcoholic group exhibited only slight deviations from the normal, and none of the cases were demented so that the intellectual capacity does not differ materially from the normal. So this fact is in absolute harmony with the clinical symptoms of the disease.

In connection with the total intellectual capacity we must consider the daily increase in the amount of work performed through practice (Übung). This is shown, first, in Chart I, now under consideration, and is represented by the lined columns at the base of the solid block columns. This is illustrated in this manner so that one can compare the daily increase with total capacity in individual cases of the three groups. And the figures given in the squares represent the values of the daily increase in each case. The values are shown graphically again in Chart 2 for a comparison of the three groups.

By comparing the values in Chart 1 it will be seen that there is considerable variation between the amount of work performed, and the daily increase through practice in different individuals. Thus, in the normal group the highest daily increase (14) occurs in two cases, in one where the total work value is 180, somewhat below the average, and in the other with a total work value of

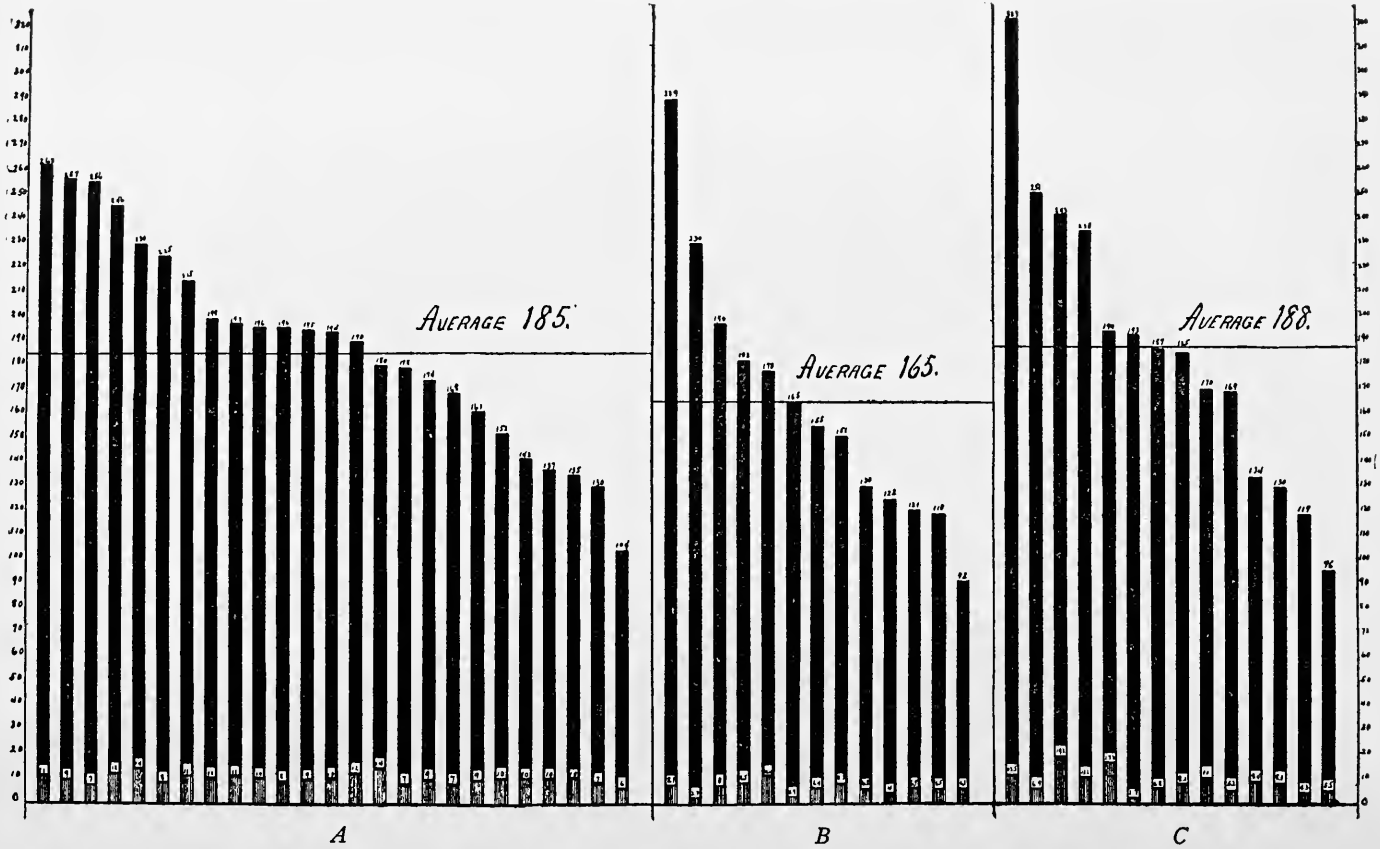


Chart I. Total mental capacity of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

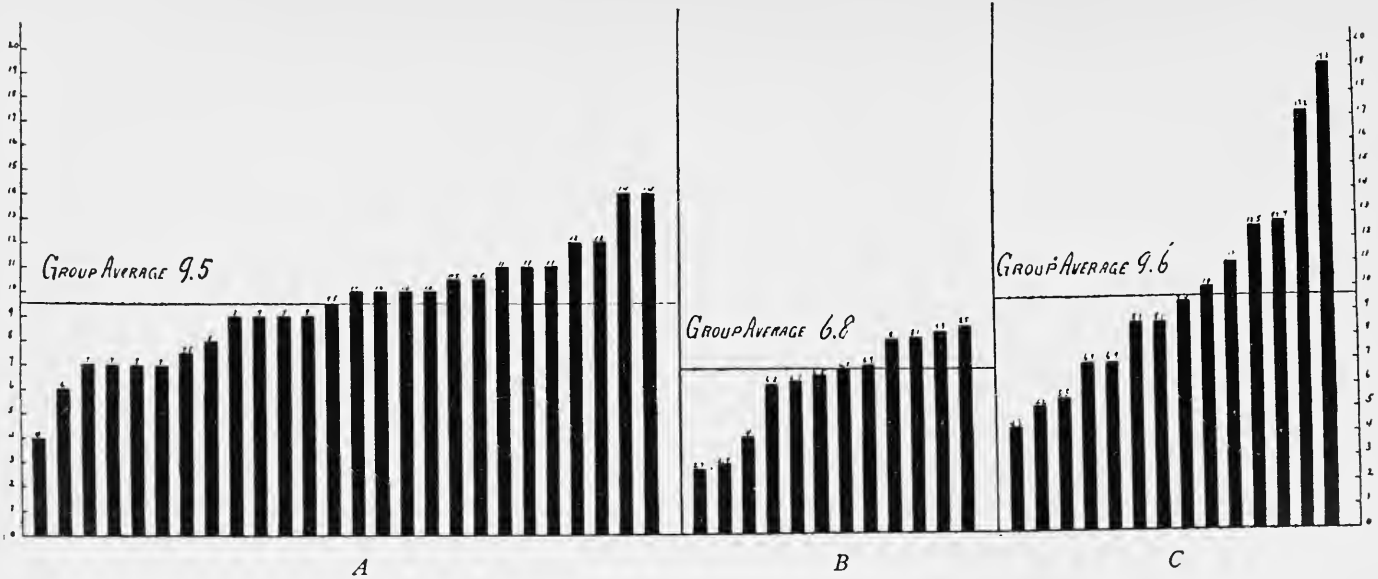


Chart 2. Daily increase of work in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

230, somewhat above the average of 185. The variations, however, are not so marked as in the dementia præcox and alcoholic groups. In the normal the average for the first 14 cases, with work values above the average for the group, is 10.2, while the average for the 11 cases below the group average (185) is 9, and by comparing Chart 2 the average daily increase is 9.5 for the normal group. Hence, in general we can state that in the normal group the daily increase is proportionate to the total work performed.

In the dementia præcox group we see more variation in the relations of these values. The lowest daily increase 2.7 is found in next the highest column, 230, and the highest daily increase, 12.4 in column 178, somewhat above the average 165. By comparing the daily increase in cases where the work values are above the average, we find almost the same condition as in the normal. The average of daily increase in five cases above the group average of work values is 8.5, which is 1.7 above the average daily increase for the group, while 6.4 represents the average of the seven columns below the group average. So that it can also be said for dementia præcox that the same conditions are demonstrated as in the normal regarding the proportion of daily increase to that amount of work done.

In the alcoholic group the variations in the daily increase show much greater variations than either dementia præcox or normal, between 19.2 and 2.7, and the average 9.5. Again considering the cases which are above the group average, we find six cases with an average of 11.6, and these below the group average in work values, eight cases with an average of 7.4. Again the rule as applied to the normal holds good for alcoholics, except that the difference between these two averages is 4.2, which is greater than either the normal or dementia præcox groups.

By comparing the three groups, as shown in Chart 2, we see that the normal and alcoholic group are practically the same (9.5, 9.6), while dementia præcox falls far below, 6.8. And this is in harmony with the facts shown in Chart 1, in which the dementia præcox group shows total capacity for work much below the normal and alcoholic group.

## Effect of Practice (Übung)

In close relation to the total intellectual capacity and average daily increase, is the effect of practice, in fact, the total amount of work accomplished and the average daily increase depend largely upon the effect of practice. The effect of practice is shown in average daily increase in the amount of work performed. In Fig. 4 we have plotted typical curves for each of the three groups. These curves are obtained by computing the amount of work performed during the first five minutes each day, and making a curve for these values for the ten consecutive days. Here, again, this second five minutes can not be utilized because of the pause as previously explained.

And for the same reason that we could not use actual figures for constructing an average work for the various groups, we could not construct a curve representing the effect of practice. Therefore somewhat typical curves from each of the groups are selected. In these curves the abscissa represents the days, and the ordinate represents total amount of work done in the first five minutes on each day. The normal curve shows a steady rise with slight variations from 70 the first day to nearly 180 on the tenth day. In other words, this individual did two and one half times the amount of work on the tenth day as he did at the beginning, or the increase was 250 per cent., due to practice and familiarity with the work. Of course, all normal curves would not show this tremendous increase, although they would approximate this curve to some extent.

The curve representing a case from the dementia præcox group, we see a decided difference. It will be noticed at first that the curve begins at a point where the normal ends, that is, the total amount of work done is much greater than in the normal case. This is shown on Chart 1, where the column marked 230 is next to the highest column of this group. But the average daily increase is only 2.7, which is the lowest of the three groups. You will notice that there is a very rapid rise for the first four days (from 170 to 275 or 160 per cent. increase), then a sudden drop to 210, and from here to the last variation, until the curve ends at 245, or 140 per cent. increase over the work done in the first minute. This shows graphically the well known clinical symptom


FIG. 4. Effect of practice in the work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

of this form of mental disease, that is, a general emotional apathy, a loss of interest in surroundings, work, pleasure, etc. So then one is not surprised to find that this feature of the disease is shown so well graphically by this method. And this curve is not the most pronounced one of the group, but is fairly representative of the group. The curve of the alcoholic patient is nearly the same as the normal, and is also fairly representative of this group, although some of the cases show more variation.

### Susceptibility to Fatigue (Chart 3)

### (*Ermüdbarkeit*)

We obtain the figures for the coefficient of fatigue, showing susceptibility to fatigue by considering the general effect of the pause upon the individual's work. This method was explained in the first part of this paper, and will not be elucidated again.

By this method we obtain the values represented by the columns in Chart 3. In the normal group we see variations in the coefficients from 1 to 17, and the average for the group is 6. It will be seen that no negative values are present, as in the dementia præcox and the alcoholic group. In the normal group, without exception, the pause or rest has had a beneficial effect upon the after work, as we saw in Fig. 3. This is not so, however, in the dementia præcox group, for in the group 8 out of the 12 cases show a negative coefficient of fatigue, varying from 5.1 to 1, and are represented by columns extending below the line 0. The average for the group is only 1. The average for the 8 negative cases is —2.8 This result is rather striking, and corresponds to the facts explained in regard to curves shown in Fig. 3, only here the comparison between the groups can be more distinctly made. It can readily be seen that the effect of the rest was very unfavorable to the dementia præcox group, and that fatigue was not present in the large majority of cases.

In the alcoholic group the average 6.5 is just above the normal and there is very little difference in the coefficients. However, we have 3 negative cases out of 14, and these 3 cases were of the dull stupid type so often seen among this group. In both groups (dementia præcox and alcoholics) the cases in which the fatigue

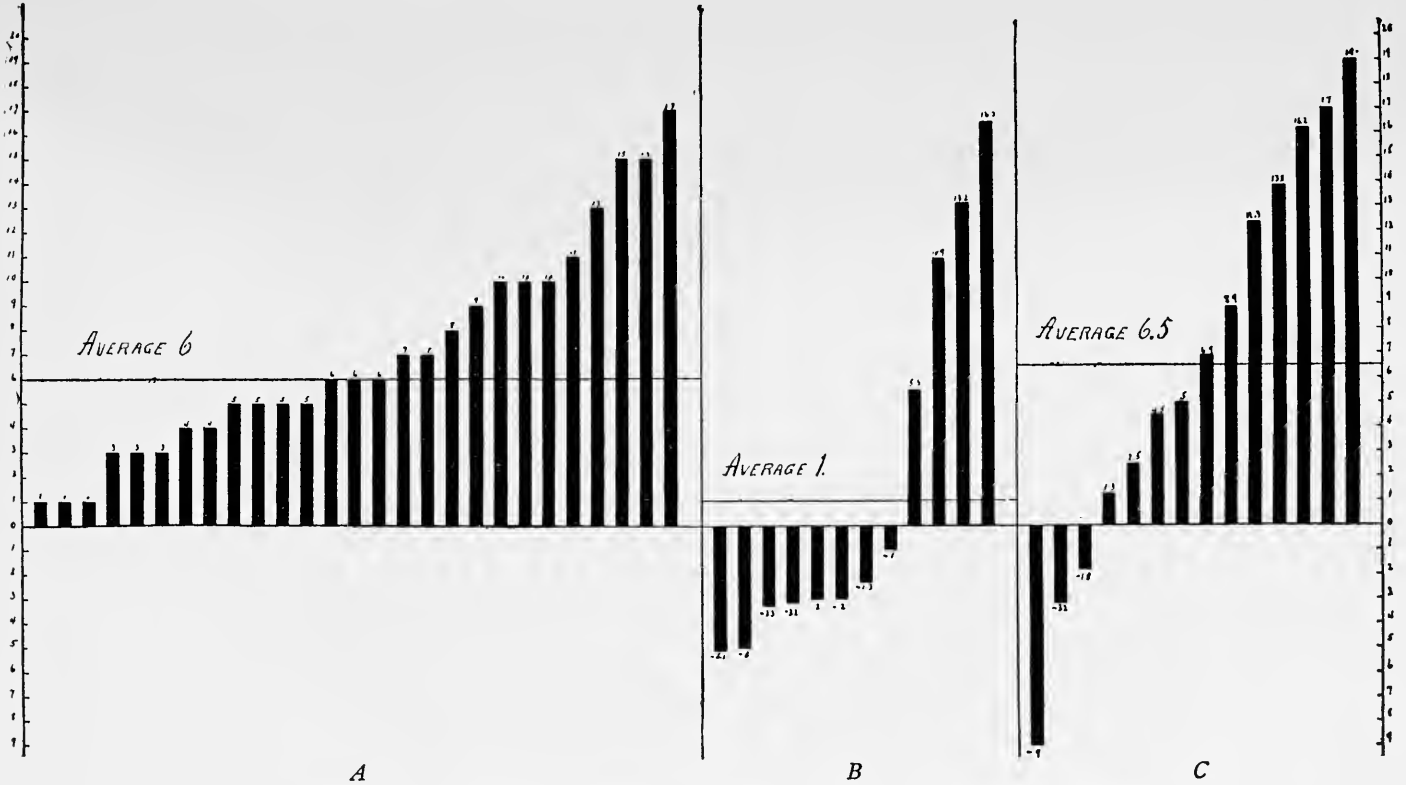


CHART 3. Susceptibility to fatigue in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

coefficient was negative, were also the cases with a smaller total capacity in comparison with other cases of the same groups.

In Chart 4 the values representing the direct effect of the pause

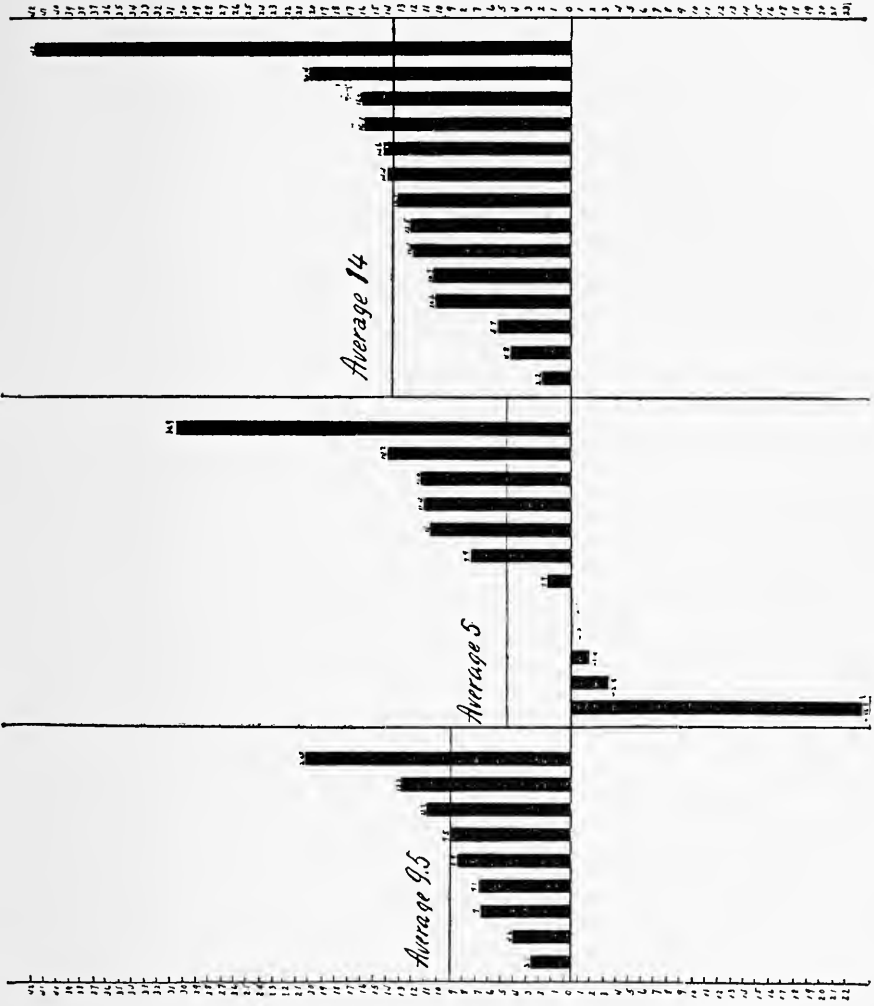


CHART 4. Direct effect of pause in work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

are shown. We have explained previously the method whereby these values were obtained. Unfortunately, I was unable to obtain in the normal group more than the values for 9 cases. But these

show no negative values. The average for the normal group is 9.5, which is considerably below the average for the alcoholic group, viz., 14. And this is also shown in Fig. 3 by the height of the dotted line after the pause where it reaches 103. And this has also been explained by the fact that in the alcoholic cases, although the fatigue values are not much above normal, yet the work after the pause shows an increase, probably because these patients after the pause have lost the effect of the work given before and start in as if they were beginning a new task. By comparing the values of the impulse after the pause, as shown in Chart 6, we find that in alcoholics the average for the group is lighter than the average for the normal, 4.5 and 1.5, respectively. And from these facts we can assume that the intensity of the will is not lost as soon as in the case of the normal group, and in consequence of this the alcoholics go to work after the pause with greater intensity of the will, and the values of the work in the sixth minute after the pause are higher than in the normal group. Also the readiness for work is lost more quickly than in the normal person, as explained above.

In dementia præcox, however, during the pause, a different process has been in operation. We have seen that no great amount of fatigue is to be compensated or dissipated by the rest, and the sudden decrease in the intensity of the will counterbalances whatever recuperation from fatigue might have taken place. Thus, the work of the last minute before the pause, and the next minute following the pause (sixth minute) does not show such a great difference as is the case in the alcoholic and the normal groups. And here again we must take into account the absence of a readiness for work, the presence of which is shown in alcoholics and in normals. For through a certain indifference to the work before them they show no anxiety to go ahead and do their best. Hence in Chart 4, five cases out of 12 are represented by negative values, and the average for the group is only 5 compared with 9.5 and 14 for the normal and alcoholic group respectively.

From these charts we can conclude, then, that the effect of the pause or rest in the alcoholic and normal groups exerted a favorable influence upon the later work, especially in the former group. But in the dementia præcox group a pause of the same length had

a very unfavorable effect upon their work. That fatigue is greater in alcoholics than in normal individuals has been shown by other methods and experiments, and the facts found here corroborate the views of other investigators.

### Intensity of the Will (Impulse)

#### Chart 5

In normal individuals the intensity of the will, or the impulse of the will is influenced by four factors, as follows: (1) In the beginning of any work the feeling is that there is something to be overcome, or some task to be performed, and that causes a high tension of the will; (2) the intensity of the will is shown at the end of a task when one wants to do as much as possible before one finishes; (3) the entrance of fatigue causes a feeling of weariness, and in order to overcome this feeling one, so to speak, strains the will to greater activity; (4) after a disturbance in a certain period of work or task, one's attention having been distracted, one begins again to work, and the impulse of the will is again shown. This is shown in the experiments by the decrease in amount of work done in the second or third minute. Comparing this with the first minute before and after the pause, we must now endeavor to find how the normal relation is disturbed in our abnormal groups.

We see that the initial impulse at the start of the task is much greater in normal than in alcoholics or dementia præcox, thus, the values are 29, 9 and 11, respectively. And when we consider that the fatigue in the dementia præcox group is very small, the fact that no great intensity of the will is present, these two facts harmonize. By observing the combined work curve, Fig. 3, we will see this explained. And if we have no marked impulse or tension of the will the only way to explain this sudden decrease in the third or fourth minute is by a sudden failure of the tension of the will. The sudden sinking of the curve to 83 in dementia præcox is in great contrast to the gradual sinking of the curve in alcoholics and normal, respectively 89 and 91. These latter figures show distinctly the effect of fatigue which has overcome the tension of the will. And we have seen that the fatigue is very small in dementia præcox and cannot come into play in explanation of the sudden sinking of the curve.

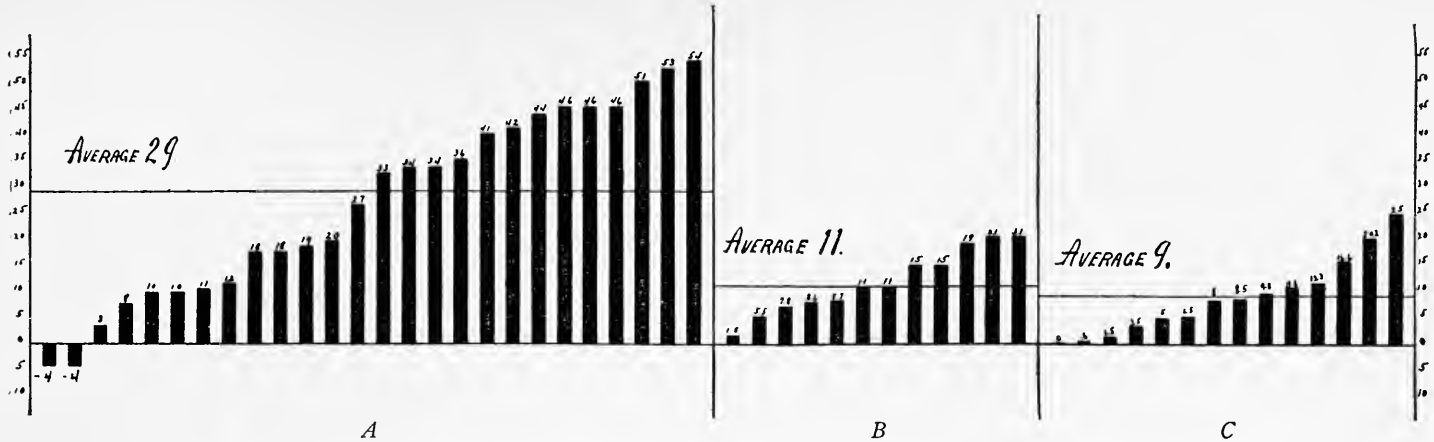


Chart 5. Intensity of the will in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

The impulse after the pause shows two facts: First, that during the pause the fatigue has been compensated in the alcoholics, and in consequence we see a tremendous rise in the curve after the pause. The curve rises to 103, which is higher than in the start of the task, and the tension of the will is not as high as at the start. How is it in dementia præcox? We have seen that we have a lesser intensity of the will, and therefore less fatigue, also a sudden sinking of the curve in the fourth minute. And we have an apparent high point of the curve after the pause, but only to 95.6, which is very much below the alcoholics. And during the pause no recovery has taken place, and the work after the pause is less than the alcoholics. Then follows again the sudden failure of the tension of the will. The pause has been unfavorable, for the amount of work done after the pause falls below the amount of work done without a pause (see curve, Fig. 3). Again we must take into account the readiness for work.

We must conclude that in both groups a rapid disappearance of the readiness for work has taken place during the five minute pause, and in normal this is explained by the fact that at the start of the work the normal person shows a great deal of interest, but after a short pause of five minutes the tension of the will has decreased considerably compared with the tension before the pause. We know from experience (although such a fact has not been established experimentally) that normal persons hold the readiness and interest for work during a short pause. And when they again begin to work they have the feeling that the task is not so difficult because they know what they have to do. Therefore, they do not exert themselves, and the amount of work done by the normal after the pause is much less than that performed by the alcoholics. Also, at the end the curve sinks, while the curve without pause rises a little bit because of practice and familiarity. And we conclude (1) that the tension of the will before and after the pause, which in dementia præcox and alcoholics has very nearly the same value, is caused by the fact that the readiness for work is rapidly lost during the pause, and they begin to work after the pause just as if they had a new piece of work to do. While the great difference of the normal will tension before and after the pause is due to the fact that this readiness

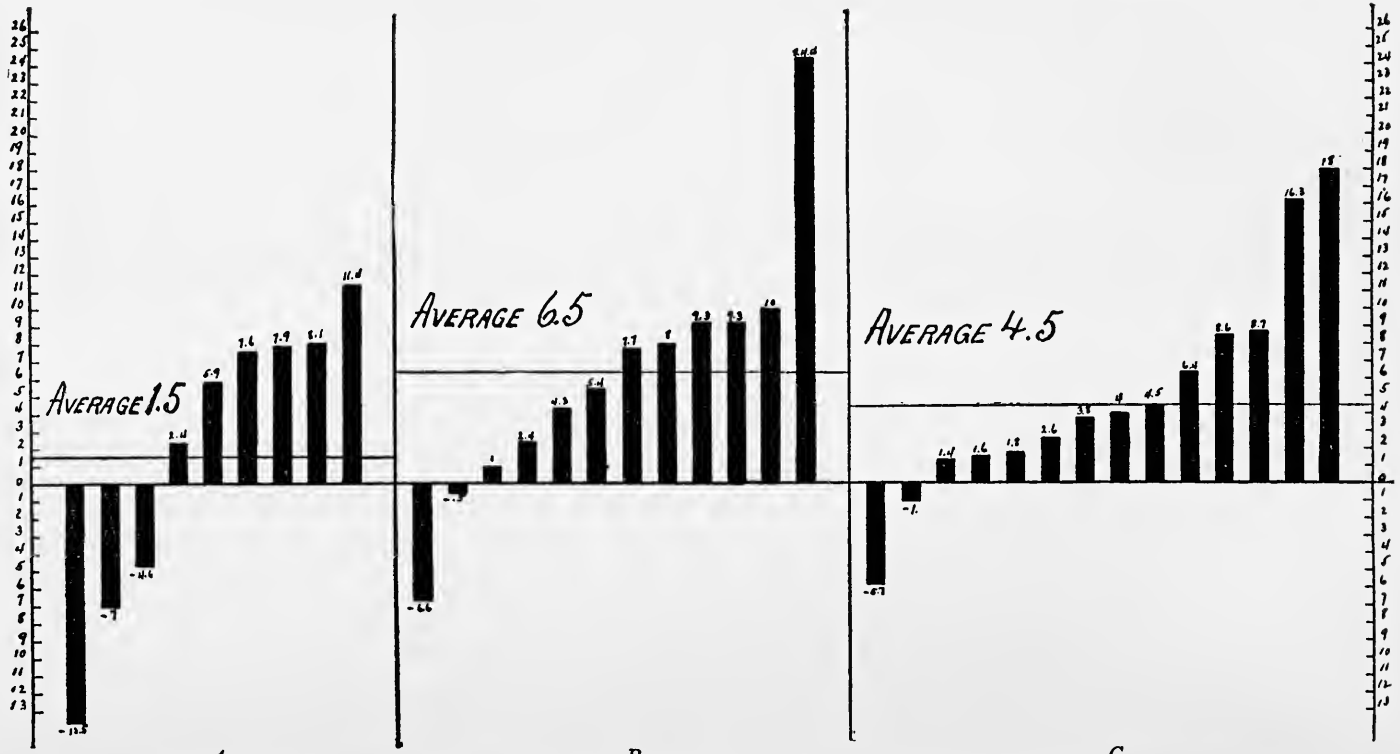


CHART 6. Values of impulse after pause in work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

for work has not disappeared, they are prepared for the task at hand.

It can be demonstrated by experiment that after pauses or rest periods of different lengths that the readiness for preparedness for work in normal also is lost, and the tension of the will is gradually increased when the time of the pause is lengthened.

*Second.* That the increase in the work done after the pause in alcoholics is due to the dissipation[4] of fatigue during the pause, and also the higher tension of the will. In dementia præcox the total capacity is not so high as in alcoholics, but the impulse of the will is about the same as before and after the pause, because no fatigue has been counterbalanced, and the readiness for work has disappeared. The irregular course of the work curve, now high, now low, shows distinctly the fluctuation of the tension of the will.

In Chart 6 we have illustrated the impulse of the will after the pause. What we have said regarding the work curves in Fig. 3 is corroborated by the facts deduced from this chart. In the normal group we have 3 cases out of 29 showing negative values, and the average for the group is only 1.5. In the dementia præcox group there are only 2 negative values out of 12 patients examined, and in the alcoholic group only 2 negative values out of 14 patients examined. The average for the dementia præcox group is here shown to be higher than the other 2 groups, or 6.5 compared to 1.5 normal and 4.5 alcoholic. And this greater value for dementia præcox as compared with the normal and alcoholics is only apparent, or when we consider the space between the sixth minute dotted line and sixth minute full line in the 3 groups. In Fig. 3 this increase is explained. It is not that the cases of dementia præcox do so much more work after the pause, or that the impulse is greater, but the sinking of the curve before the pause due to the neglect of the will to act, accounts for this light average in dementia præcox.

## Conclusions

By the simple experimental procedure outlined in this paper, we have shown:

1. That the disturbance of the will is the most important symptom of Dementia Præcox.

[4] Erhohlung.

2. Because of this defect of the will, the rest from work, so beneficial to normal individuals, and the alcoholic cases, has an unfavorable influence in Dementia Præcox.

3. The effect of practice in Dementia Præcox is of much less value than in normal persons and alcoholic cases, which conforms to the general apathy shown in Dementia Præcox cases.

4. Absolute deflection of the will, a prominent symptom in Dementia Præcox, is shown by this method.

5. Fatigue is absent in Dementia Præcox, and greater in alcoholics than in normal persons.

6. Absolute mental capacity, as shown by the amount of work performed, varies but little in the three groups, being less in Dementia Præcox than in the other two.

7. That the daily average increase in the individual cases is proportionate to the total amount of work performed, is true of the three groups.

8. That the experimental results agree with the clinical symptoms of Dementia Præcox and Alcoholic Insanity.

# THE RELATIONSHIP OF HYSTERIA, PSYCHASTHENIA, AND DEMENTIA PRÆCOX

By Adolf Meyer, M.D.

PROFESSOR OF PSYCHIATRY, JOHNS HOPKINS UNIVERSITY

A report of two cases furnishes the material for our discussion; one of undoubted hysteria in which a catatonic dementia præcox supervened; and a case of psychasthenia with obvious deterioration. Unfortunately the material for a discussion of the present status of the conceptions of hysteria, psychasthenia, etc., would prove too long even if I should limit myself to giving the main facts, in view of the tremendous confusion that exists owing to the ease with which programmes and definitions are launched, and owing to the fact that as a rule the contentions are made without documentation by actual records. To my regret I also must abstain from the publication of my two cases in full, in the form in which alone they would be wholly convincing.

Suffice it to say that the first case presented an hysterical hip for one year at 11; a classical hysterical paraplegia with convulsions for one year at 21, another four months of hysterical convulsions and spinal and ovarian hyperesthesia at 23; finally at 28, a psychosis of the character of hysterical delirium, with a relapse which passed into a simple stupor; after one year the patient was submitted to an aggressive thyroid treatment, passed into an exhausting excitement, and after a few months into a classical catatonic stupor lasting several years and relaxed but gradually. Each of the above steps set in on the ground of decided strains; the development was fairly consistent and the residual after about fifteen years since the first outspoken psychosis, a condition of nursing a "pain" or a "disease" in the spine, stereotyped manners, soliloquies in imaginary settings, but in the main a slow response to efforts to reëstablish a certain adaptability to her home environment and to simple home interests.

The second case is that of a young man of 27, as a boy an indiscriminate reader and imaginative, slightly odd at college, with a growing obsession of incompleteness of his toilet, the brush-

ing of the teeth and the drying after the bath—a feeling of dampness, a desire to rub himself dry—incompleteness and indecision are the patient's own words for his condition. The ruminative episodes have become more and more automatic and they dominate the patient's life with undeniable dilapidation of all capacity of application and interest.

In teaching we can present the first case as one showing that plain hysteria does not protect against dementia præcox, or is not incompatible with it, and more, namely, that the mechanism can become progressive.

The second case shows that the simple deterioration may carry with it the picture of psychasthenia, feeling of incompleteness and indecision and automatism, and practically no other trait, under which conditions we must nevertheless treat the disorder like one of psychasthenia.

The relative inaccessibility of both cases makes it difficult to reconstruct the whole picture of evolution as well as was done in Dr. Hoch's cases. But they are suggestive at least as a foundation to the question of the relation of hysteria, psychasthenia and dementia præcox.

For any discussion of such a relation we should review what we would accept as safe ground concerning the entities. From the point of view of teaching it would be desirable to keep the three entities clearly apart if that agrees with the facts. From the point of view of constructive work and analysis, it is better to make the most, not necessarily of the identification with some dogmatic compound picture which is sure to be open to unwarranted twists by extraneous factors, but rather of the working factors or determining conditions that constitute the deviation in specific cases, and the modifiability of the conditions. This constructive method is available in terms of reaction-types rather than of disease entities.[1]

The issue is a demand for safety and clearness in using the facts at hand, a demand to work with what the case actually presents rather than through identification with a compound picture which we do not see and have to take on authority, with inevitable varieties of definitions (I merely refer to Dr. Dana's recent effort

[1] Compare the Problems of Mental Reaction-Types, Mental Causes and Diseases, Psycholog. Bulletin, Vol. V, 245–261.

to limit hysteria and to create other types). The synthetic method is also more apt to disclose factors worth knowing for prophylaxis, and can be taught as readily and efficiently for work as the other, although perhaps not as easily for examinations.

The issue then is what reaction is at work? What are the determining conditions? What is their modifiability? What are the alternatives of prospect and their determination?

In this respect the Freud-Jung conceptions command most attention, but they are to-day largely emphasis of a portion of the situation. My personal preference is for a broad formulation of the problems in terms of substitutive activity.

All biological function is an adaptation which demands for its safe expression a statement of (1) the determining conditions, (2) the form of the reaction, and (3) the result or terminal condition.

Among the adaptative disorders which occupy us we do well to try and make a fairly clean-cut distinction between simple insufficiency such as we see in imbecility and those disorders which tend to be progressive and cumulative. Simple insufficiency may make impossible certain reactions, but need not expose the individual to destructive false attempts. The borderland between imbecility and positive substitutive reactions begins where the individual does not merely fail to react but uses poorly planned and ill-adapted make-shifts, such as tend to undermine the development or maintenance of healthy instincts.

We deal in the first place with a great number of individuals with non-systematized constitutional inferiority, forming the borderland between imbecility and the essentially pathological substitutive reaction types.

The next level of reaction is the neurasthenic which according to Dejerine and Moebius may be looked upon as the common source of the other neuroses, or if you wish to put it the other way, the prodromal type of many further developments. The hypochondriacal reaction type would be closely connected with it; the psychasthenic with its ruminations and obsessions and automatisms and panics would form another branch. The hysterical reaction type with its submersion and conversion into most distinct examples of substitutive reactions, forms the group which more than any other occupies the physician because it shows

us clearly the involvement of the physical component of our mental activity.

Psychology is but reluctantly coming to size up its data in terms of conduct and behavior, with due attention to the actual physical component. We find further the merely dilapidating or distinctly incongruous reaction types marking the tendency to deterioration; the scattered fantastic ruminations or the tense catatonic types, or the sham consistency of the paranoic development. Quite a few paranoic conditions belong to this group, whereas the manic-depressive reaction type would in many instances remain more or less doubtful in its position and seem to be more akin to the not clearly psychologically determined epilepsy series of physiological fluctuations. As all biological terms, such a conception as substitutive activity has its specially appropriate *centers* of utility and it shades off into domains where it is less useful. It is plain enough in the psychasthenic and hysterical symptoms which are plainly substitutions for what is dodged.

One common method is to speak of distinct "diseases," and I, without mentioning the term "disease" especially, would indeed speak of "pure cultures of reaction types," of cases in which a special reaction type is dominant, and of cases in which it is incidental or subordinate. The terms which stand for the formula of specific diseases are of didactic importance and especially justified, because some of the reactions practically exclude one another as is the case between hysteria and psychasthenia, whereas, other combinations are compatible and produce all those transition forms which resist definition as units. In any case a definition should contain the essence of the mechanism. Dr. Dana speaks of the disease hysteria as being a morbid mental condition in which ideas or emotional stress seriously and unwittingly control the body and produce more or less permanent and objective morbid states,—and he then uses as the protype those cases produced by railroad accidents, shocks and collisions and alcoholism; i. e., cases in which the non-psychic nervous disorders are evidently very predominant and with them the frequently bad prognosis: whereas most of the other hysterias are classed with the psychataxias or psychasthenias as not true hysteria. It ought to be plain that unless we make identification with a name the main issue, it is better to train the student to think

of pure and impure or complicated reaction types rather than of "true" hysteria, psychasthenia, etc., making a disease of an arbitrarily limited group of cases, without evidence that the fundamental definition explains these cases and with serious reasons to assume that really, matters not mentioned in the definition give the group its character. It is especially undesirable to accept a set of nosological units put forth without the documental material of records to back up the contentions as is the case with Kraepelin.

With these principles in mind I reserve the term dementia præcox to the essential deteriorations in which there is absolutely no doubt about the deterioration, and give some descriptive term to the reaction type at hand, hysterical, psychasthenic, hebephrenic, catatonic, paranoid, or whatever designates the mechanism as long as deterioration is not in the center. I do this with all the more justification if I consider that Kraepelin in his book says that among the admissions to a clinic the dementia præcox cases form but a slightly higher percentage than the manic depressive; whereas Wilmanns on the same material from Heidelberg reports that between 1901 and 1905 the rate of dementia præcox varied between 40 and 52 per cent. of admissions, and the manic-depressive ranked between 11 and 16 per cent. The question thus arises very seriously—is it right to swell the issue of the probable or possible outcome to such importance as to become the leading conception of the disease? This idea is in the ascendency just now. Two years ago Janet told me that annually a number of hysterical patients were transferred to the insane wards with deterioration; last summer he felt inclined to yield to the tendency to mark these cases outright as cases of dementia præcox. In his descriptions of psychasthenia, however, he shows a number of cases of progress to deterioration similar to my second case. Peterson and Jung refer to the fact that there are many cases of dementia præcox which for years are not to be distinguished from hysteria and that a large number of catatonic processes were formerly called degenerative hysterical psychoses. Jung's analysis shows very clearly the common factor in the developments of a hysterical and of a deterioration reaction. If he speaks of the stabilization of the complex he gives a plain expression of the plain fact; but he yields to the temptation of accounting for this

stabilization, etc., by toxines, a method which to my mind leaves too readily the functional ground on which the whole investigation has prospered and the plain facts are exposed.

I thus come to the next point of the discussion; that is, what constitutes the unfavorable reaction type? As I pointed out in a paper read before this society over three years ago, and again in the Toronto discussion[2] we have strong reasons to consider the foundation for these reaction types to be the result of conflicts and deviations of instincts, and in cases of deteriorations we find invariably that the complex-phenomena occur in an especially vulnerable field, or denote from the start the deficiency of balancing instincts.

Why should a patient drift so strongly into more or less absurd imaginations pre-eminently in the sexual domain, in religious elaborations and in fantastic spheres? Why should there be such a striking tendency to ideas of reference which denote so strongly a feeling of inferiority of action? What determines the striking tendency to feelings of passivity, of being influenced, etc., in the automatisms, which the more hysterical takes in a self-possessed, not in a passive attitude? A careful study of the cases shows the ravages of habitually incomplete or directly inadequate and ill-adapted and ill-controlled reactions, a tendency away from the contact with reality and self-correction, a scattering of the personality, with or without the sham consistency which we see in the paranoic forms, and through it all a stultifying of the instincts which are essential for balancing in the complex demands of life. We, therefore, attribute a pernicious effect to a trauma in the sense of Freud and Jung, even when, instead of its merely leading to the hysterical reaction type, it plays a rôle in an actual interference of development of instincts. In this respect it takes little skill to realize how different the sexual evolution of the hysterical is from that of the dementia præcox case; there is much more inferiority or miscarriage of function in the future dementia præcox case, to suggest voices or electric currents in the womb, or the imaginations of love answered by a stranger through passive movements of the tongue. In all this we invariably see additional interweaving of habit-deteriorations, ruminations instead of youthful pranks and of a rash trial and

[2] British Medical Journal, September, 1906.

rejection method of the more wholesome development, with its instinct for touch with reality. And beside these defects we see precocious one-sided moralizing, top-heaviness, leading the patient further and further away from the life with concrete corrections.

The effect of the spreading into ill-protected domains and the determination of the seriousness of such combinations is easy to demonstrate in the question of masturbation, which to this day is dealt with in the most dogmatic and absurd fashion by a large number of physicians who think they can settle the issue by the negation of all importance, and the mere assumption of fundamental defect where it happens to lead to disaster. Masturbation, like the use of alcohol, must be judged specially in every case.

It will be the task of a publication of a sufficient number of thoroughly studied cases to show the lines of cleavage between deviations of instincts which do and others which do not tend to become progressively destructive. To think of these matters in terms of auto-intoxication is not ruled out if such a relation is demonstrated, nor should we of course be satisfied with an abstract statement that we deal with conflicts and deterioration of instincts; but at the present juncture it is best to recognize the probability that in many cases a number of factors combine and that among these many can only be expressed in activities, habits and instincts and that a sweeping over-simplifying terminology obscures the clearness of observation and reasoning.

Neurology has led us too much out of a functional appreciation of developments. It reasons largely with stationary and progressive focal conditions and their occasional repair, rather than with balancing mechanisms, such as we must work with in psychopathology.

The conception of substitutive reactions brings us back to a normal foundation of direct activity again. It frees us from excessive definitions at the loose end, furthers definition of the actual situation and of the means of adaptation available in the patient. Instead of a plan of identification with names of arbitrary patterns we get attention to the facts at hand without arbitrary expurgations. We can see and teach what we have in the pure cultures of hysteria, psychasthenia, and dementia præcox, and if we find collaboration of special factors, we have a place

for them according to the facts, be they of the type of regulation of conduct and behavior, i. e., mental, or infrapsychical, toxic or what not.

This mode of presentation can be made just as simple as that which works with disease-entities and much more just and espeially much more valuable in plans of handling the case and in shaping facts for prophylaxis.

# AN EXPERIMENTAL STUDY OF THE OCULAR REACTIONS IN THE PSYCHOSES, FROM PHOTOGRAPHIC RECORDS

By Allen Ross Diefendorf, M.D.

lecturer in psychiatry in yale university medical school

and Raymond Dodge, Ph.D.

professor of psychology in wesleyan university

Within the last few years exact knowledge of the normal movements of the eyes has made rapid advance, particularly in America, through the use of mechanical and photographic registering devices. Quantitative information is now at hand with respect to the angle-velocity (2, 3, 9)[1] and the path of the line of regard during rapid eye-movements (4–6, 8, 10–16),[1] the accuracy, stability and variability of fixation under a considerable variety of circumstances (4, 6, 8, 10–15),[1] the ocular reaction-time (8),[1] and the peculiar modifications of eye-movements which constitute short-lived motor habits (5),[1] pursuit movements (7),[1] coordinate compensatory eye-movements (7, 8),[1] and the movements of convergence (12).[1]

The ease with which the photographic technique can be adapted to a wide variety of experimental requirements, together with certain peculiarities of the eye-movements themselves, led the writers to believe that a comparative study of the eye-movements of normal and insane persons might be made a fruitful contribution to our experimental knowledge of the reactions of the insane.

Such a comparative study might well find its basis in any of a large variety of experimental data. The present investigation was limited to three main problems, which were relatively clear to us, and to meet which we framed our technique.

The difficulties in any experimental study of normal psycho-

[1] Numbers 1 to 16 refer to the bibliography at the end of the paper.

physical processes are serious enough, even though one may rely on the highest degree of intelligent coöperation on the part of the subject. In the study of abnormal mental life, additional difficulties arise from the very nature of the object of investigation, in direct proportion to its variation from the normal. As Kraepelin cogently observes, lack of comprehension of the experimental test, lack of ability to execute it, lack of interest, coöperation, and of endurance, all conspire to increase the task of the experimenter and to modify the value of his results. The consequent demand for trustworthy experimental methods, which, without too complicated technique or too unusual demands on the patient, shall yield quantitative information of significant variations from normal reactions, voices at once the need and the embarrassment of experimental psychiatry. These demands are met in part at least by the reactions of the eyes as they are known through their photographic records.

Eye-movements are neither unusual nor difficult. On the contrary, the ability to look at a bright object which appears suddenly within the field of view is one of the earliest forms of motor organization to be achieved, and it is retained long after the ability to learn new and complex forms of reaction is irretrievably lost. A patient will naturally look at a suddenly appearing object when he could learn the simplest new reaction only imperfectly and with enormous waste of time.

Moreover, while the eye-movements are intimately associated with the most complex mental activities, they are peculiarly inaccessible to subjective observation. Even the best trained observers utterly failed by introspection to discover some of the facts of eye-movements which are most conspicuous in the photographic records. For the ordinary man, his eye-movements are usually involuntary and unconscious; they are essentially a part of the mechanical preadjustments of vision, and consciousness is concerned with the result rather than the preadjustments. They may on occasion be consciously initiated, but once begun they are entirely withdrawn from conscious control. This effectually prevents both arbitrary modifications and successful simulation of pathological symptoms. Moreover, relative uniformity in the previous training gives unequalled opportunity for legitimate comparison between different persons. Probably no other form

of reaction is common to so many different persons in so high a state of development. The nearest competitors in this respect are the various speech functions; but gross differences of individual habits and training render generalization concerning the latter more difficult. In the eye-movements, on the other hand, we may reasonably assume almost complete identity of practice and a general high grade of efficiency.

Finally, the technique of recording the eye-movements is relatively simple. By using the corneal reflection as the registering medium, there is absolutely no discomfort to the patient and no unusual stimulus to excite him, while the real purport of the experiment may be entirely hidden under the instructions to the patient to read exposed figures, letters, or short words, or simply to try if he can see anything at all on the black screen.

On the other hand, the photographic procedure is not without some difficulties of its own. The eyelid may droop and interfere with the recording light without parallel interference with vision. Excessive head-movements may render a considerable portion of the plate illegible or take the patient out of focus of the recording camera. But the records themselves are their own vindication, and we venture to believe that those who can appreciate the experimental difficulties of securing trustworthy comparative data will find some satisfaction in our results, and reasonable ground for expecting more of the general procedure in the future.

The most serious limitations of our technique arise from defective vision. Just how far this may finally be found an embarrassment it is difficult to predict, since the technique permits some use of correcting glasses. The series of experiments we undertook was planned to render a considerable range of visual defects indifferent; nevertheless, in three cases we were compelled to abandon the tests because of gross refractive errors.

For both of us the most surprising feature of the experiment was the conduct of the patients during the tests. We anticipated a considerable variety of troubles, particularly from the maniacal patients, and safeguarded the apparatus in a number of entirely unnecessary ways. Naturally the worst cases of maniacal excitement were not requisitioned; but, as will later appear in detail, we succeeded in getting excellent records from patients that in the wards appeared utterly impossible.

In only two cases did we encounter any unwillingness to participate in the tests. Something about the experiments seemed to appeal to the patients. The majority were helpful in getting into position and maintaining it. Many seemed thoroughly to enjoy the sessions; some were interested in the results; some were more sensitive than others to the blue light, as was evidenced by an occasional increased tendency to wink or to withdraw entirely from the apparatus, but the light was stopped down by blue glass so that continuous fixation produced only a mild and inoffensive after-image. Many of the patients apparently failed to notice it.

## Apparatus and Technique

The registering device for recording the eye-reactions was a modification of the Dodge photochronograph which was designed by one of the authors, and has been used by him practically in its present form during the last five years.

As used by us it consisted of an enlarging camera of fixed length (about 5 ft. [153 cm.]) fitted with a Bausch and Lomb convertible protar, Series VIIa. The device for producing a regular motion of the sensitive photographic plate was the Dodge-Cline falling plate-holder. It consists of a light-tight box 2 ft. (61 cm.) high and 7 in. (17.8 cm.) wide, fitted with opaque slides, and capable of quick adjustment to the rear of the enlarging camera like a regular plate-holder. Within the box a movable frame, holding a 5 in. (12.7 cm.) by 7 in. (17.8 cm.) photographic plate, slides vertically on two brass tracks, so adjusted that all lateral play is taken up by springs. The movement of the sliding plate frame is accurately controlled by a simple hydrostatic device. The frame is attached to a piston-rod ending in a plunger which works in a vertical cylinder of lubricating oil. The valve of the plunger offers no resistance as the latter is raised through the oil, but it absolutely resists every effort to force it downwards. The release of the plate is effected by opening a stop-cock below the plunger, when the weight of the plate-holder forces the oil out of the cylinder at the bottom through the stop-cock, and returns it to the top of the cylinder above the plunger. The velocity of the fall is determined by the opening of the stop-cock, the viscosity of the oil, and the weight of the frame and plate.

The photographic record is made on the falling plate by photographing the image of an electric arc as it appears at the cornea of the eye. The rays of the arc light are first passed through blue glass screens to eliminate the photographically useless but physiologically disturbing rays of the lower spectrum. Those rays which are then reflected from the convex surface of the cornea to the camera are brought to a focus on the photographic plate by the lens of the enlarging camera.

A convenient and well-nigh necessary modification of the plate-holder was introduced for the first time in the present experiments. Since one cannot presuppose much coöperation on the part of the insane in finding and maintaining the proper position of the head, it was found impossible to rely on methods of focusing that were satisfactory for normal subjects. A focusing and finding glass was consequently introduced into the falling plate-holder just below the photographic plate, and in the same plane. A system of automatic screens was arranged, so that during the focusing process the plate was entirely protected from light. In this way we could quietly await the opportune moment and quickly adjust the apparatus to changes in the patient's position. The apparent movement of the corneal reflection is slightly less than half the actual movement of the eyes.[2]

But the actual displacement is magnified by the enlarging camera so that the total record is a continuous curve whose amplitude is about three times the actual amplitude of horizontal eye-movements, and whose height is determined by the velocity of the plate. Under the above circumstances, continuous visual fixation will be recorded as a straight vertical line on the falling plate, while any horizontal movement of the eyes will be indicated by an oblique line whose obliquity depends on the relative velocity of the horizontal and vertical components.

The difference in illumination between the corneal reflection of the arc light and its background is sufficiently marked, so that on a perfect plate there is no trace of other impressions except the record made by the corneal image of the arc light. This should appear as a fine black hair-line on an almost clear back-

[2] For the mathematical theory of the movements of the corneal reflection and empirical tests of its accuracy as a measure of eye-movements, see Dodge, Experimental Study of Visual Fixation (8).

ground. No limit has been reached in the number of records one plate will hold, except the purely mechanical confusion of the lines.

In our experiments the stimulus was so arranged as to begin coincidently with the beginning of the record. When the velocity of the plate is known, the duration of an eye-reaction will be given by the height of the vertical line of eye-fixation between the beginning of the record and the beginning of the oblique line of eye-movement. The velocity of horizontal eye-movement should be given by the obliquity of the line of eye-movement, i. e., the time, as measured by the fall of the plate, between one fixation and its successor.

Two series of records were made. One depended for its time-record on the measured length and obliquity of the lines. In the other an interruption of the recording light by a tuning-fork gave the time directly in vibrations of the tuning-fork. Notwithstanding a complex system of controls, our first records of the angle-velocity of the eyes by the former method involved such serious sources of error that we were reluctantly constrained to abandon them as practically useless. They do not appear in this report. The reaction-records, on the other hand, were about equally satisfactory in both series. Each record involves a probable error of less than 0.01 second.

In convenience of control and in general trustworthiness the tuning-fork interruption of the recording light is a decided advantage. It was arranged as follows:

An arc light, with horizontal upper carbon, was mounted on a heavy optical bench behind a large condensing lens. In front of this lens, and at the point where the latter brought the rays of the arc light to a focus, was placed an opaque screen with an opening which was so shaped and oriented that at each vibration of an electric tuning-fork (a tested Koenig tuning-fork of 100.12 c.-p. per second) the opening was alternately opened and closed to the passage of the light from the arc. A second smaller lens of 6 in. focus was so placed as to render the transmitted rays parallel. From the position of the subject one isolated vibration of the fork exposed the arc light and cut it off again. With this interrupted light, when the tuning-fork was in continuous vibration, each record of the corneal reflection appeared on the slowly falling

photographic plate as a line of black points or dashes. From the beginning of one dash to the beginning of the next represented a time interval of 0.01 second. The duration of any fixation or of any eye-movement could be read directly from the appropriate record in units of 0.01 second by counting the corresponding dots.

The arclight and the tuning-fork interrupter were placed in one corner of the laboratory at a distance of about 15 ft. (459 cm.) from the patient. The patient was seated comfortably at the apparatus just in front of the enlarging camera. His head was held as firmly as practicable against a side-rest and a nosepiece.[1] Further constraint seemed inadvisable. The resulting records were considerably complicated by head-movements, but our immediate interest was not a study of the spatial characteristics of the eye-movements, but rather a study of their temporal succession. For this purpose the records were unequivocal, except in a few cases. No patient was in the apparatus more than thirty minutes. Under favorable circumstances the tests occupied about fifteen minutes, including periods of relaxation. All records are for monocular vision. A black cardboard screen completely hid the unused eye. Three groups of tests were made on each patient at each sitting, and all three groups were recorded on the same

[1] *Figure 1,* a half tone, could not be inserted here. It is a picture of the entire apparatus set up and shows on the left the subject comfortably seated in a chair with the arms resting on the table and the head placed against a side, top and nose rest. About one foot in front and on a level with the eyes there is a single photographic lens fitted in the end of an enlarging camera box five feet long with an appropriate standard and terminating in the box of the falling plate holder, all resting on the same long table. On the extreme right and fifteen feet distance from the subject on another table is the light apparatus with appropriate hood, lenses and colored glass screen to produce the desired rays, so thoroughly stopped down as not to lighten the darkened room and in front of these the black screen with the Koenig tuning fork, resting on the same table. Partly hidden by the camera box, resting on the same table with it one and one half feet in front and a little to the left of the patient is shown two large black screens with openings for the arc recording light. These screens are as nearly as possible in the same plane, the rear one being movable and carrying the test objects for exposure through the openings in the front one. Immediately in front of this is swung the pendulum, which, when not in use, is hitched up out of sight of the subject.

plate. In this way each plate made a complete experimental record of a single patient at the time of examination. The plates were carefully numbered, and each number was entered in a permanent record against the name of the subject, with such additional notes of the clinical picture and conduct of the patient as seemed pertinent.

The experiment which we undertook included three tests for distinct but inter-related phenomena.

### 1. Velocity of Eye-Movements

#### (A) *Theory*

The first test concerned the angle-velocity of simple reactive eye-movement. Experiments on normal individuals have shown a remarkable uniformity in the angle-velocity of similar uninterrupted eye-movements of the same person, quite independent of direct conscious effort to move the eyes fast or slowly. There are slight variations of the two eyes, and slight variations in successive movements, but under similar circumstances these variations are relatively small. The first published records of the angle-velocity of the eye-movements noted a slight but clear slowing up of a rapid succession of eye-movements toward the end of a series of ten movements. This was tentatively attributed to fatigue. A series of records taken in connection with a hitherto unpublished study of fatigue confirmed the previous findings and justified the hypothesis that retardation of the velocity of the eye-movements is a phenomenon of fatigue.

Valuable as they undoubtedly would be, it was hardly to be expected that adequate fatigue tests could be obtained from the insane. On the other hand, it seemed plausible that the different disease processes, in so far as they affected the psychomotor processes at all, would variously affect the angle-velocity of the eye-movements. It also seemed probable that such variations in a type of movement which is equally practised for all subjects and is almost entirely removed from the effects of voluntary caprice, would furnish exceptionally trustworthy comparative data.

Naturally, our immediate interest centered in patients suffering from maniacal-depressive insanity, where, as a matter of fact, the

most marked variations from normal velocity were found, but the results of the test in other disease-processes are not without interest.

### (B) *Experimental Conditions*

The test for the angle-velocity of the eye-movements necessitated the experimental production of a considerable number of rapid reactive eye-movements of the first type (Dodge [7]) of approximately the same amplitude. Taking advantage of the fact that rapid eye-movements separate the fixation-pauses (or moments of clear vision) in reading, we satisfied the experimental requirement by exposing a succession of isolated numerals in two different parts of the field of regard about 25° apart. The reading of one numeral by the patient was the signal for the operator to cover it and to expose another 25° from the former. When the latter was read, it in turn disappeared and another was exposed where the first had been.

The eyes rarely moved through the entire 25° from numeral to numeral in a single rapid eye-movement. This was entirely congruent with the known facts that practically every long eye-movement involves more or less final readjustment in the form of short corrective movements. When the object of interest is relatively obscure, like a numeral 25° from the fixation-point, the normal end-corrective movements will vary from 1° to 5°. In the great majority of cases the corrective movement is in the same direction as the initial movement. This indicates that the initial movement was too short. If the corrective movement was negative, or if it exceeded one-sixth of the total displacement, the record was discarded. The average main corrective movements in our accepted records is about 3°. This reduces the average displacement corresponding with our records to about 22°.

A large black screen was placed at 18 in. (45.75 cm.) in front of the subject, at one side of the camera. This screen was permanent and served all three experiments. It was pierced by three openings in the same horizontal line. Two openings for the exposure of objects were 8 in. (20.4 cm.) apart. The middle opening for the passage of the blue recording light was $5\frac{1}{2}$ in. (14 cm.) from the left hand opening and was lost in the blind spot of

the right eye when the center of the left hand opening was fixed. This arrangement with respect to the blind spot was designed to lessen distraction by the light during the preliminary focusing of the camera. A movable black cardboard screen behind the permanent screen carried a series of numerals. These were so oriented that as the screen fell, step by step, the numbers were successively exposed at the appropriate openings of the fixed screen. The movements of the screen were regulated by the operator. The signal to the operator was the reading of the exposed numerals by the subject. The amplitude of each movement of the screen was automatically regulated by an appropriate escapement.

Before each series of experiments two numbers were exposed respectively in the left and right hand opening, the middle one being closed. The patient was told that other numbers would appear in the same places, and that these were to be read aloud as rapidly as possible. Foreigners were encouraged to use the most familiar language. With the initial movement of the screen the middle opening was uncovered, allowing the subdued arc light to illuminate the subject's eye. Four groups of these movement-records were taken for each subject, making, when all the lines were legible, twenty-four movements. This number was unnecessarily large, since the mean variation is regularly less than half of the unit of measurement, but a tendency to coördinate winking just at the time of eye-movements together with head-movements and interrupted eye-movements made some of the individual records useless.

Since the illumination of the subject's eye was the condition of a photographic record, the simultaneous exposure and illumination were a mechanical guarantee that the beginning of the photographic record was synchronous with the appearance of the stimulus to eye-movement. This arrangement gave the chief instrumental condition for the second and third series of experiments to determine the reaction-time of the ocular movements.

### (C) *Results*

Table I shows the average duration of eye-movements of *cr.* 22 for nine normals, twenty-one maniacal-depressives, in both the maniacal and depressive phases, four cases of dementia præcox

of the hebephrenic type, four epileptics, six paretics and one imbecile.

Under each form of insanity the data are arranged according to the severity of the disease. The most marked cases come first. Each case is described at length under the corresponding number in the Appendix. All time-values are given in $\frac{1}{1000}$ second.

TABLE I

| Normal | Mania[3] | Depression[4] |
|---|---|---|
| | *Marked* | *Marked* |
| 1 ..... R. D. ..... 59 | 13 ... S. S. ..... 59 | 26 .... H. N. (*a-c*) 55 |
| 2 ..... A. ..... 59 | 14 ... J. C. ..... 44 | 32 .... P. F. (*a*) .. 74 |
| 3 ..... Wh. ..... 61 | 15 ... W. B. ..... 60 | 29 .... R. R. ..... 77 |
| 4 ..... Wr. ..... 60 | 18 ... T. S. ..... 51 | Average ..... 69 |
| *Female Nurses* | 19 ... J. G. ..... 55 | |
| 9 ..... Ll. ..... 60 | Average ..... 54 | *Less Marked* |
| 10 ..... E. F. .... 54 | | 33 .... K. B. ..... 72 |
| | *Less Marked* | |
| *Male Nurses* | 31 ... L. K. (*b*) .. 60 | *Slight* |
| 11 ..... W. S. .... 62 | 21 ... A. H. ..... 56 | 34 .... T. B. ..... 51 |
| 12 ..... J. R. .... 60 | 27 ... M. G. (*c-d*). 47 | High .... 77—marked |
| | 33 ... K. B. (*b*) .. 59 | Low ..... 51—slight |
| *Recovered Patient* | Average ..... 55 | |
| 17 ..... M. D. (*c*). 60 | | |
| Average ..... 59 | Hypomania | |
| Low ..... 54 | 22 ... G. ..... 50 | |
| High ..... 62 | 29 ... R. R. (*a*) .. 65 | |
| M. V. ..... 1.5 | 23 ... A. T. ..... 76 | |
| | 20 ... S. K. (*c*) .. 51 | |
| | 24 ... P. B. ..... 61 | |
| | 25 ... P. R. ..... 70 | |
| | Average ..... 62 | |
| | Low .....44, marked | |
| | High .....76, hypo | |

| Dementia Præcox | Dementia Paralytica | Epileptic |
|---|---|---|
| *Marked* | *Marked* | *Moderate* |
| 36 ..... A. R. ..... 62 | 43 ..... D. D. .... 54 | 51 ..... G. L. ..... 67 |
| 37 ..... M. B. ..... 52 | 44 ..... W. H. .... 53 | 52 ..... J. F. ..... 64 |
| | 45 ..... A. B. .... 60 | 53 ..... H. O. .... 67 |
| *Moderate* | 47 ..... A. S. ..... 60 | 54 ..... M. B. .... 80 |
| 40 ..... G. L. ..... 60 | Average ..... 57 | Average ..... 69 |
| 41 ..... M. F. ..... 50 | | |

[3] Maniacal phase of manic-depressive insanity.
[4] Depressed phase of manic-depressive insanity.

| *Slight* | *Less Marked* | Imbecile |
|---|---|---|
| 42 ..... B. M. ..... 56 | 48 ..... J. P. E. ... 54 | 57 ..... R. H. .... 60 |
| | 49 ..... J. A. ..... 49 | |
| Average ..... 56 | Average ..... 51 | |

From the preceding table (I) it is obvious that the velocity of the eye-movements of manic-depressive patients does not vary exactly with the degree of depression or of maniacal excitement which they present. On the other hand, it should be noted that there is some variation even among normal persons. In any fair evaluation of our data, then, one must allow at the outset for some individual variations, independent of all disease. The origin of these individual variations is at present a matter of conjecture. Earlier studies (6, 9) demonstrated that the differences between individuals are not absolutely constant for different angles of movement, or for the same angle of movement at different times. But these variations are relatively small, and are due, in part at least, to minor variations in the action of opposed and coöperating muscles which are not further analysed, and which may be grouped together for our purposes as chance variations. But after due allowance is made for these chance variations, the grossness of the variations in the insane and certain very obvious tendencies in different diseases and in different phases of the same disease indicate some causal interdependence with the disease itself.

While it would be injudicious to regard these tendencies as settled before our data have been materially increased, the marked variations of the extreme maniacal and the extreme depressive states may safely be regarded as characteristic. This appears not merely from Table I, but still more convincingly from the history of such cases as 26, H. N., p. 173; 29, R. R., p. 173; and 33, K. B., p. 173. So again both patients suffering from dementia præcox and dementia paralytica have abnormally rapid eye-movements, while the epileptics are notably long. The slow eye-movements of the extreme depressive and the quick eye-movements of the extreme maniacal coincide with the general phycho-motor disturbances as they appear in the familiar clinical picture of these psychoses. Quantitative evidence of abnormal quickness of

maniacal movements has, however, hitherto been conspicuously lacking. On the other hand, it is again congruent with the

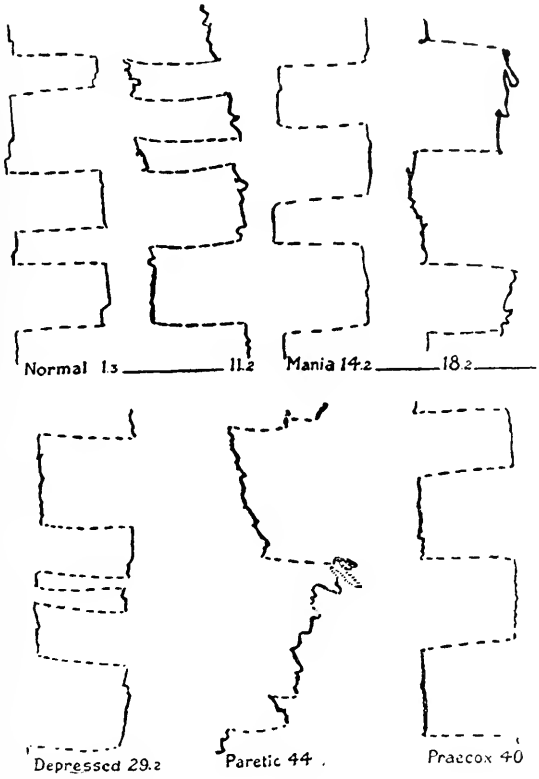


PLATE I

Plate I is a reproduction of typical records of eye-movements. The records were projected by lantern and drawn from the projected image on a much enlarged scale. These drawings are here reprouced by process on a somewhat reduced scale. The resulting lines reproduce the original records very well, save that the dashes are relatively fainter grey in the records. The exact shape of each dash is not accurately reproduced. In all cases which are represented in Plate I the photographic plate was moving so slowly that the dots run together in the vertical lines, appearing as dashes only during eye-movement. The dashes represent flashes of light succeeding each other every 0.01 second. The paretic line, No. 44.1, is an extreme case of head-movement and broken lines. The broken movements are typical, the head-movements less so.

disease-picture that the eye-movements, which we have found to be rapid, are secondary automatic acts, not those that require conscious direction and control such as have hitherto been measured; and it certainly corresponds closely with our general knowledge of the diffusion of the sensory impulses and the interaction of the higher and lower nervous centers, that these secondary automatic movements should reach their extreme velocity when the interaction of the higher nervous centers is lessened.

The slowness of the eye-movements in the depressives and in the epileptics cannot be accounted for conversely by excessive interference of the higher centers. It seems rather to be the expression of a more widespread involvement resulting in a general inefficiency of the whole psychomotor system and including not only the higher centers, which appeared to be chiefly involved in mania, but also the lower centers, the simple reflexes, and the automatic acts.

Besides the mere differences of velocity in the eye-movements, there are certain characteristic tendencies in form and accuracy of eye-movement that our technique was not designed to measure, but which may be mentioned in passing. Along with the increased velocity in the maniacal eye-movements, there is a parallel tendency to abnormal overshoots such as were first described in normal persons by E. B. Huey (3). Depressive eye-movements are more regular and symmetrical. The eye slides up into the new position as though against a gradually increasing resistance.

The eye-movements of the grossly demented show marked inaccuracies of fixation. Advanced dementia paralytica has curious inconsequential fixations, breaking the normal eye-movements at irregular points.

### 2. Ocular Reaction-Time to New Peripheral Stimuli

The second test concerned the simple reaction of the eye in responding to a peripheral stimulus. Like the first test, the second also depended on the regular and usually wholly unconscious habit of fixing a numeral or letter one is expected to read.

#### (A) *Experimental Conditions*

The procedure was as follows: A figure 6 was exposed in the left hand slit of the permanent screen. The subject's attention

was directed to it with the instructions that other figures, which would appear at one side or other of the 6, must be read as rapidly as possible. With the final warning to look sharp, the 6 suddenly dropped out of sight and 1 in. (2.5 cm.) to the right or left there appeared a different numeral. The exposure apparatus resembled that used in the first series of experiments. A special exposure screen was prepared so as to expose one figure (6) at the center of the left hand opening in the permanent screen when the exposure screen occupied its primary position. A series of numbers was pasted on pieces of black cardboard which could be slipped into place either to the right or left of the 6 and just so far above it as would bring them into view by one stroke of the escapement. It only required $\frac{1}{2}$ in. (1.25 cm.) movement of the exposure screen to carry the 6 out of sight and to expose the new number. The movement was so rapid that it seemed like an instantaneous change. Without the appearance of motion in any direction, the one seemed to disappear and the other was in place. The same movement of the exposure screen uncovered the arc light and began the photographic record. Simply counting the dashes of which the record was composed from the beginning of the record to the beginning of the eye-movements to fix the new number gave the reaction time of the eye in 0.01 second. A similar experiment is described more in detail in Dodge's "Experimental Study of Visual Fixation" (8). Vocal reaction to printed matter or to isolated words, such as Dodge studied, seemed inexpedient in these tests on account of gross differences in education. Four ocular reactions were taken for each subject. Unfortunately, in some cases extreme head-movements made some of the records uncertain, while winking made other records useless. These two disturbances combined materially to reduce the number of available records.

The small number of reactions for any one individual is a serious limitation to the use of our data. If we had the work to do over again, we are agreed that we should venture to increase the number of simple reactions. Our reason for limiting the number in the present tests was the consciousness that we were dealing with subjects who were abnormally susceptible to fatigue of attention. To some of them even four tests of the same

kind seemed many. For the sake of comparison, we ran through a series of ten reactions each with two more tractable cases. The results show that, in these two cases at least, the smaller number did no violence to the facts. In later discussions it will appear that minimal reactions and the general variability are quite as important as the rather meaningless averages.

## (B) *Results*

### TABLE II

SIMPLE OCULAR REACTIONS TO PERIPHERAL STIMULI

| 1. NORMAL | 2. MANIA | 3. DEPRESSION |
|---|---|---|
| | *Marked* | *Marked* |
| 1 ... R. D. ... 200 | 13 .... S. S. ..... 205 | 26 ... H. N. (*a*) 250 |
| 2 ... A. ..... 200 | 14 .... J. C. ..... 210 | 27 ... M. G. ... 295 |
| 3 ... Wh. ..... 215 | " ..... 217 | 29 ... R. R. (*c*). 173 |
| 4 ... Wr. ..... 210 | 16 .... M. M. .... 225 | |
| Average ..... 206 | 17 .... M. D. (*a*).. 250 | *Less Marked* |
| | 18 .... T. S. ..... 250 | 32 ... P. F. ... 379 |
| 5 ... H. ..... 192 | 19 .... J. G. ..... 210 | |
| 6 ... T. ..... 249 | 20 .... S. K. (*b*).. 229 | *Slight* |
| 7 ... Wi. ..... 140 | Average ..... 224 | 34 ... T. B. .... 293 |
| 8 ... C. ..... 247 | | 17 ... M. D. (*b*) 204 |
| 9 ... Ll. ..... 223 | *Less Marked* | |
| 10 ... E. F. .... 195 | 31 .... L. K. (*c*).. 260 | Average ..... 266 |
| Average ..... 208 | 21 .... A. H. ..... 215 | High ..... 379 |
| | 27 .... M. G. (*d*).. 257 | Low ..... 173 |
| 11 ... W. S. .... 198 | 33 .... K. B. (*b*).. 219 | |
| 12 ... J. R. .... 225 | Average ..... 238 | |
| Average ..... 211 | | |
| | HYPOMANIA | |
| 17 ... M. D. ... 222 | 22 .... G. ..... 230 | |
| Average ..... 222 | 23 .... P. A. ..... 225 | |
| | 20 .... S. K. (*c*).. 202 | |
| Average ..... 209 | 24 .... P. B. ..... 170 | |
| High ..... 249 | 25 .... P. R. ..... 230 | |
| Low ..... 140 | Average ..... 211 | |
| | | |
| | Average ..... 224 | |
| | High ..... 260 | |
| | Low Hypo ..... 170 | |

4. Dementia Præcox

*Marked*

36 ... A. R. .... 240
37 ... M. B. .... 185
38 ... R. B. .... 258
Average ..... 228

*Moderate*

39 ... A. McI. .. 276
41 ... M. F. ... 220

*Slight*

42 ... B. M. ... 152

Average ..... 222
High ..... 276
Low ..... 152

5. Dementia Paralytica

*Marked*

43 .... D. D. ..... 370
44 .... W. H. .... 237
45 .... A. B. ..... 225
47 .... A. S. ..... 237
Average ..... 267

*Less Marked*

48 .... J. P. E. ... 190
49 .... J. A. ..... 217
Average ..... 203

*Remission*

(50 ... F. A. .... 193)

Average ..... 246
High ..... 370
Low ..... 190

6. Epileptic

*Moderate*

51 ... G. L. ... 298
52 ... J. F. ... 228
53 ... H. O. ... 195
54 ... M. B. ... 197

Average ..... 229
High ..... 298
Low ..... 195

## (C) *Discussion of Table II*

The simple ocular reaction-time is long. According to our records the normal average lies above $200\sigma$. In strict accuracy this average is undoubtedly too high, and should be reduced by a constant instrumental error of about fifteen. This error is involved in the form of the exposure of the peripheral stimulus. We have not tried to correct it, since it applies equally for all subjects, and our interest lies mainly in comparative rather than absolute time estimations. But after all corrections are made, these records agree with all the available data, and the simple ocular reaction-time is long.

One might *a priori* have expected that a reaction which is at once so common and apparently so necessary to the welfare of the individual in the conduct of life would be short. On the other hand, it must be noted that each ocular reaction to peripheral stimuli involves a considerable sensori-motor elaboration of the stimulus. The adequate reacting eye-movement is not only in a definite direction, but it is also of definite extent. The accuracy of the eye-movement does not now concern us, since we measure in reaction-time only the beginning of the reactive movement. But the beginning of every eye-movement is really only the initial phase of a movement of definite direction and extent. Before

the eye starts, the elaboration of that particular motor impulse must be relatively complete. An accurate account of the correspondence between reaction-time and reaction-accuracy is a desideratum.

In a sense, every ocular reaction to a peripheral stimulus is not a simple reaction at all, but an individual adaptation to a change in the environment. In the past, such a reaction would have borne the misleading name of a "choice reaction." The length of the simple ocular reaction, then, is not an anomaly. It corresponds directly with the complex but automatic elaboration of the sensori-motor impulse.

Abnormal reactions may result from an indefinite number of changes within this complex sensori-motor process. This is at once the inspiration and the danger of every interpretation of complex reactions. In view of the possible complications, the relatively small mean variation for normal subjects points to a relatively stable normal oculo-motor systematization. It emphasizes at the same time the gross variations of the insane.

The small number of *per capita* records forces us to consider the reactions, as we were led to consider the velocity of movement by groups rather than by individuals. Furthermore, we will again limit our generalizations to such gross variations as are inexplicable on the basis of chance variations.

The most conspicuous comparative feature of the results is the abnormally long reactions of the maniacal-depressive patients. Not only do they average long, but, with one exception, the average reactions of both the extreme and the less marked maniacal, and of all the depressed with one exception, are above the normal. These data are not novel. They agree with the reaction experiments of Franz.

In view of the unequivocal testimony of the averages, it is somewhat disappointing to note that there is no direct correspondence between the duration of the ocular reactions and the clinical judgment of the severity of the disease. It is hardly an accident that in spite of the high averages in cases of mania the most extreme maniacal excitement had the shortest ocular reaction; while the maniacal group, which averages the longest, is that of the less marked excitement. In view of the complication of the reaction-process and the number of unanalyzed factors,

we feel that any hypothesis of the effect of the disease on the reaction must be regarded as tentative. But on grounds which will appear most clearly in the discussion of the pursuit-reactions, we believe that the inconsequential reactions of maniacal excitement are due to opposed tendencies in the inter-relation of the superior and the secondary central systematizations.

### 3. Ocular Pursuit-Reactions

#### (A) *Theory*

The third series of tests was a reaction experiment of unusual character. The simplest and, in the end, also the most complex ocular reaction with which we are acquainted is the pursuit-movement in reaction to moving pendulum.

It is the simplest, in the sense that no new object of regard is furnished as stimulus for reaction. There is no change in the object of attention. An object is fixed, and the fixation lapses through the movement of the object fixed. The reëstablishment of the lapsed fixation involves a form of ocular reaction such as occurs on an average several times a minute throughout the waking day, either because the object moves or because of involuntary displacement of the eyes by bodily movements. Pursuit-reactions, as we may call them, normally involve a reaction-time slightly longer than the simple reactions to peripheral stimuli. At least one factor tending to lengthen the pursuit-reaction appears directly in the form of the stimulus. The stimulus to reaction is not given in the release of the pendulum, but only when, after release, the pendulum has moved some appreciable distance. The amount of movement that will constitute a stimulus to pursuit will depend on the training of the subject and the accuracy with which he maintains his fixations.

Paradoxical as it might at first seem, the total pursuit-reaction finally involves more extensive psychomotor elaboration than any other ocular reaction that we know how to produce. As is now well known (Dodge [7]), the true pursuit eye-movements are totally different in function and character from the rapid reaction-movements of the eyes by which peripheral objects of interest are fixated. The rapid movements are relatively constant in duration, and they constitute moments of practical blindness.

The reason for this eye-movement blindness is still under discussion. There is no debate concerning the fact. The pursuit-movements, on the other hand, vary in angle-velocity with the angle-velocity of the moving object. The eyes move fast or slowly as the object moves fast or slowly. Moreover, the pursuit-movements are pre-eminently moments of relatively clear vision. It is because we wish to see an object clearly that we move the eyes as the object moves and keep its image on retinal areas of relatively clear vision. Furthermore, the psychomotor elaboration of the simple ocular reaction is fixed by the long-established habit of bringing excitations of the peripheral retina to areas of clear vision. The psychomotor elaboration of the pursuit-movements, on the other hand, is in practically each instance of pursuit an unique psychomotor problem. The reaction to pursuit is fixed and habitual enough, but the velocity of the eye which shall correspond to the velocity of the object at the distance it chances to be can scarcely ever be a motor habit. To be a successful pursuit there must be an adaptation of the general pursuit tendency to the peculiar condition of each separate instance. Especially in the pendulum pursuit-movements are these conditions so various as to present, in each new case, practically unique conditions. Angle-velocity, apparent amplitude, and period of oscillation would all be alike only if pendulums of the same length swing through the same arc at the same distance from the eye of the observer. Yet unique as each case actually is, a normal eye will fall into an adequate pendulum pursuit-movement with surprising quickness and accuracy. In every normal individual the very first fixation after the initial reaction to a moving pendulum has the characteristic true pursuit-slide, even though it usually corresponds in angle-velocity to the first part of the pendulum swing, and is consequently too slow. Let us emphasize the fact: for normal individuals, however inadequate the first attempt to fix the moving object may be, it always has the characteristics of a true pursuit-movement (see Plate, lines 1, 10, 11).

The return swing of a second pendulum is usually followed with precision, except at or near the middle of the arc of oscillation, when one or two short, sharp, rapid movements break the simple pendulum pursuits. The character of these pendulum pursuits scarcely alters, even after a large number of experiments

under the same objective condition. Each new pursuit seems to be solved de novo, and the short-lived motor habits involved in every adequate pursuit seem to be lost when the pursuit is interrupted (8).

This ability to elaborate adequate pursuit-movements, i. e., to adopt an adequate motor response to the peculiar situation presented by the rhythmic movements of an object, varies widely in mental disease. In some respects, the most marked variations are found in the pendulum pursuit-movements in dementia præcox, where a marked hesitation to fall into the swing of the pendulum was found even in the mildest cases. While this peculiarity is apparently not absolutely restricted to dementia præcox, it was found in other patients only where the disease-process has produced marked deterioration.

### (B) *Experimental Conditions*

The instrumental device for producing the pursuit-reaction and the subsequent pursuit-movements was a number attached to the bob of a second pendulum. The latter hung just in front of the fixed screen with its axis vertically above the middle point between the extreme left and right hand opening. Before the experiment the pendulum was held out of equilibrium in front of the left hand opening by a simple catch attached to a falling screen in the usual place behind the fixed screen. This falling screen was released by the operator, as in the other experiments, and the release of the screen simultaneously started the pendulum and opened the way for the recording beam of light. In every case the patient was previously shown how the pendulum moved and was then requested to watch the number closely, to keep his eyes on it, not to lose it, watch it, &c.

## (C) *Results*

### TABLE III

OCULAR REACTIONS IN PURSUIT OF A MOVING STIMULUS

#### 1. NORMAL

1 ... R. D. .... 198
2 ... A. ..... 233
3 ... Wh. ..... 243
4 ... Wr. ..... 210
Average ..... 221

5 ... H. ..... 274
6 ... T. ..... 284
7 ... Wi. ..... 216
8 ... C. ..... 225
9 ... Ll. ..... 265
10 ... E. F. .... 285
Average ..... 258

11 ... W. S. .... 235
12 ... J. R. .... 223
Average ..... 229

17 ... M. D. ... 223

Average ..... 239
High ..... 285
Low ..... 198

#### 2. MANIA

*Marked*

13 .... S. S. ..... 190
14 .... J. C. ..... 225
" ..... 222
16 .... M. M. ..... 343
18 .... T. S. ..... 217
19 .... J. G. ..... 225
20 .... S. K. ..... 314
Average ..... 248

*Less Marked*

31 .... L. K. (*c*) .. 260
21 .... A. H. ..... 217
27 .... M. G. (*c-d*) 305
33 .... K. B. (*b*) .. 255
Average ..... 259

HYPOMANIA

22 .... G. ..... 210
29 .... R. R. (*a*) .. 220
23 .... P. A. ..... 240
20 .... S. K. (*c*) .. 212
24 .... P. B. ..... 155
25 .... P. R. ..... 273
Average ..... 218

Average ..... 240
High (marked).... 343
Low (Hypo)..... 155

#### 3. DEPRESSION

*Marked*

27 ... M. G. .... 379
28 ... M. C. .... 255
29 ... R. R. (*c*). 230
30 ... L. W. ... 339
Average ..... 301

*Less Marked*
*Slight*

34 ... T. B. .... 303
17 ... M. D. (*b*) 272
Average ..... 287

Average ..... 296
High ..... 379
Low ..... 230

#### 4. DEMENTIA PRÆCOX

*Marked*

36 ... A. R. .... 305
37 ... M. B. .... 195

*Moderate*

39 ... A. McI. .. 207
40 ... G. L. .... 260
41 ... M. F. .... 215

*Slight*

42 ... B. M. .... 275
Average ..... 243
High ..... 305
Low ..... 195

#### 5. DEMENTIA PARALYTICA

*Marked*

43 .... D. D. ..... 445
44 .... W. H. ..... 240
45 .... A. B. ..... 224
47 .... A. S. ..... 267

*Less Marked*

48 .... J. P. E. .... 227

Average ..... 281
High ..... 445
Low ..... 224
Average excluding high ..... 239

#### 6. EPILEPTIC

*Moderate*

51 ... G. L. .... 280
52 ... J. F. ..... 222
53 ... H. O. .... 230
54 ... M. B. .... 221
Average ..... 238

## TABLE IV

TABLE OF COMPARISON OF AVERAGES

| NORMAL | | | DEPRESSION | | | DEMENTIA PARALYTICA | | |
|---|---|---|---|---|---|---|---|---|
| | | | *Marked* | | | *Marked* | | |
| Mvt. | Reactions | Pursuit-reactions | Mvt. | Reactioss | Pursuit-reactions | Mxt. | Reactions | Pursuit-reactions |
| 59 | 209 | 239 | 69 | 239 | 301 | 57 | 267 | 294 |
| MANIA | | | | | | | | |
| *Marked* | | | *Less Marked and Slight* | | | *Total* | | |
| 54 | 224 | 248 | 61 | 295 | 287 | 55 | 246 | 281 |
| *Less Marked* | | | DEMENTIA PRÆCOX | | | EPILEPTIC | | |
| 55 | 238 | 259 | *Moderate, Marked and Slight* | | | 69 | 229 | 238 |
| HYPOMANIA | | | 56 | 222 | 243 | | | |
| 62 | 211 | 218 | | | | | | |

### (D) *Discussion of Table III*

In all classes except in moderate depression the pursuit-movement reaction averages longer than the simple ocular reaction. The differences between the two, however, are not constant. Even in the group of normal persons the differences are not constant. The variations, however, allow of some degree of classification. It must be remembered that the stimulus to pursuit-movement is not mechanically fixed as was the stimulus to a new peripheral stimulus. The movements of the pendulum operate as a stimulus to pursuit only when the lapsed fixation in some way makes itself felt through the indistinctness of the object. For the trained observer it operates almost immediately. For the less trained it operates only after the pendulum has moved some clearly appreciable distance. The mean difference between pendulum- and pursuit-reactions for normal observers under the conditions of our test was approximately $30\sigma$. This average difference holds approximately the same for præcox and for the longer reactions of paresis. It does not hold for maniacal-depressives. But the individual variations are so great that the maniacal-depressive differences can scarcely be spoken of as characteristic. It does, however, constitute additional evidence of a high degree of disturbance of those complex superior central processes which are usually grouped under the general name of attention. Further evidence to the same effect comes from a consideration of minimal reactions, which are given in Table V.

## TABLE V

### Table of Minimal Reactions

| Normal | | Mania | | Depression | |
|---|---|---|---|---|---|
| 1 | 180 ..... 180 | Mania | | 26 ..... 200 | |
| 2 | 180 ..... 200 | 13 ..... 180 | ..... 180 | 27 ..... 279 | ..... 244 |
| 3 | 206 ..... 230 | 14 ..... 180 | ..... 180 | 28 ..... | ..... 228 |
| 4 | 200 ..... 200 | 16 ..... 186 | ..... 257 | 28 ..... 170 | ..... 210 |
| 5 | 166 ..... 260 | 17 ..... 205 | | 216 | ..... 227 |
| 6 | 230 ..... 273 | 18 ..... 210 | ..... 200 | 34 ..... 240 | ..... 300 |
| 7 | 128 ..... 179 | 19 ..... 160 | ..... 210 | 17 (*b*).. 186 | ..... 248 |
| 8 | 243 ..... 179 | 20 ..... 182 | ..... 257 | 213 | ..... 274 |
| 9 | 200 ..... 240 | 186 | ..... 214 | Average 215 | ..... 246 |
| 10 | 180 ..... 260 | 31 ..... 230 | ..... 240 | | |
| 11 | 140 ..... 200 | 21 ..... 190 | ..... 210 | Epileptic | |
| 12 | 210 ..... 210 | 27 ..... 230 | ..... 280 | 51 ..... 270 | ..... 250 |
| 17 | 220 ..... 190 | 33 ..... 190 | ..... 190 | 52 ..... 200 | ..... 200 |
| Average 191 | ..... 216 | 210 | ..... 230 | 53 ..... 190 | ..... 210 |
| | | 22 ..... 210 | ..... 200 | 54 ..... 170 | ..... 200 |
| | | 23 ..... 210 | ..... 200 | Average 207 | ..... 215 |
| | | 20 ..... 190 | ..... 200 | | |
| | | 24 ..... 150 | ..... 140 | | |
| | | 25 ..... 210 | ..... 220 | | |
| | | 194 | ..... 192 | | |
| | | Average. 195 | ..... 211 | | |

| Dementia Præcox | | Dementia Paralytica | |
|---|---|---|---|
| 36 | ..... 200 ..... 260 | 43 | .....350 ..... 290 |
| 37 | ..... 180 ..... 190 | 44 | ..... 220 ..... 200 |
| 38 | ..... 248 | 45 | ..... 210 ..... 210 |
| 39 | ..... 260 ..... 167 | 47 | ..... 230 ..... 240 |
| 40 | ..... ..... 240 | 48 | ..... 170 ..... 210 |
| 41 | ..... 200 ..... 180 | 49 | ..... 180 |
| 42 | ..... 150 ..... 230 | 50 | ..... 180 |
| Average 206 | ..... 211 | Average. 220 | ..... 230 |

*Discussion of the Table of Minimal Reactions (Table V)*

In some respects the minimal reactions for any well-established type of reaction is more instructive than the average reaction. The minimal reaction shows the reflex systematization in its highest state of efficiency. The mean reaction indicates the average state of efficiency. The two differ from one another by the mean value of all those disturbing elements that may complicate the reaction-process. It seems to the writers a very significant fact that the average minimal reaction of extreme mania-

cal excitement is below the average minimum of normal subjects. It is not much below the normal, but it is not above it as the total average is. Moreover, the mean variation of the minimal reactions is approximately the same as the mean variations of the averages. This consistent uniformity is not accidental. In connection with the adequate pursuit-reactions it seems to the writers to constitute unequivocal evidence that the oculo-motor systematization is not seriously disordered in acute mania. The large mean value of disturbing elements constitutes the final point that we have to offer in the cumulative experimental evidence that extreme mania involves a marked disturbance of the controls normally exercised by the superior central systematizations.

This seems to the writers to coincide closely with the general clinical picture of marked mania. The motor organization even for complex acts is not lost. The incapacity for regular employment is flagrantly due to gross disturbances of the normal controls within the higher systematizations. Tentatively, at least, we may picture this in terms of an inhibition of the free interaction of the various factors in the normal complex superior organization.

Provided there is some intrinsic retardation of the intermediate systematizations, like the simple oculo-motor reflexes, we should expect to find the total evidences of maniacal excitement less marked. In such cases we should expect the minimal reactions to be long as well as the average reactions. This is actually the case in less marked mania. It looks as though the inhibitory processes involved in the disease were affecting lower centres. The climax of this downward progression seems to be reached when, in *extreme depression,* the resistance to neutral interaction involves the simplest reflexes.

In contrast to the differential increase of resistance to neural activity as found in the manic-depressives, our experimental data from the demented point to a general disorganization of the central systematizations.

In dementia paralytica the entire nervous system is involved in this disorganization, as is shown by the marked retardation, and the inefficiency of the simplest, as well as of the higher reflexes (patellar, pupillary, and cerebellar).

In præcox the disorganizations seem to be primarily limited to

the superior systematizations. This is shown negatively by the rapid eye-movements, normal oculo-motor reactions, and positively by the difficulty of adopting adequate reactions to new conditions of the environment, as in the pursuit-movements. This latter peculiarity of præcox patients has a practical as well as a theoretical interest.

Practically, it is an important differentiating symptom between moderate maniacal excitement and developing præcox, i. e., between two psychoses whose differential diagnosis is of the utmost importance and often of the utmost difficulty. Unfortunately the faultiness of the pursuit is not easily detected by direct observation. Photographic registration, although remarkably simple as a scientific technique, is rather too expensive in time and apparatus for regular professional use. If the matter prove worth while, a simplified recording apparatus is probably the only safe and practicable solution. It would seem strange, however, if similar phenomena cannot be found in other forms of reaction which are more accessible to direct observation.

The theoretical bearing of the inadequate ocular pursuit-movements of præcox we have already mentioned. It was not overlooked by us that the simplest explanation of the phenomenon would be to coördinate it with those processes which are ordinarily grouped under the head of faulty attention. This explanation seems to us untenable on the following grounds:

1. Equally grave defects of "attention" exist in maniacal excitement without parallel difficulty of pursuit.

2. The reaction-times do not indicate gross defects of "attention" in moderate præcox.

3. Reasonable "attention" and effort at pursuit are both clearly indicated in the number and character of the short corrective movements.

Finally, the phenomenon seems to connect itself naturally with certain characteristic clinical observations of præcox as one manifestation of the patient's inability to adapt himself to new and unusual requirements of his environment. Put technically, it is the patient's inability to adopt adequate short-lived habits in response to a new recurrent situation. There is some clinical evidence that this motor phenomenon rests on a basis of faulty elaboration of the perceptual data. Our experiments indicate

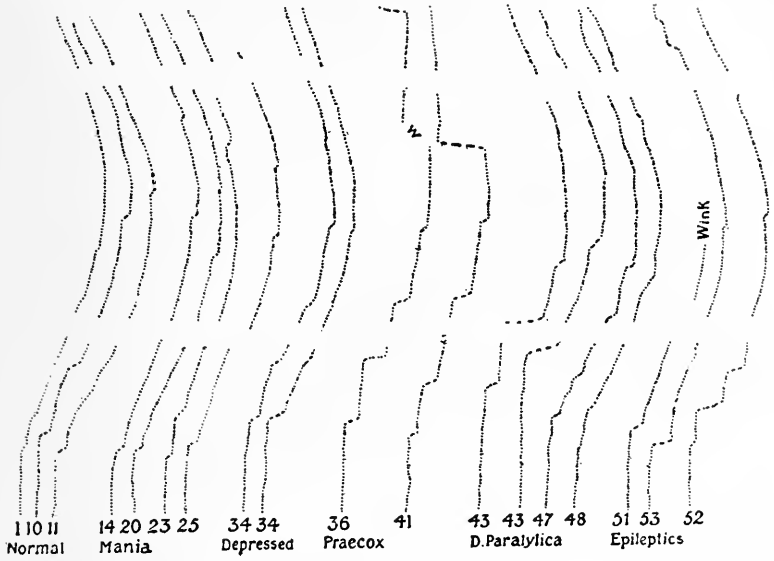


PLATE II

Plate II is reproduced from drawings of typical pursuit-reaction records. It shows most of the typical variations of the visual pursuits so far as they were not complicated by gross head-movements. The lines accurately reproduce only the general configuration of the pursuit.

Each line, reading from the bottom up, represents one complete pursuit-swing corresponding to a double oscillation of the second pendulum. Since the release of the pendulum and the beginning of the record are synchronous, the straight line at the beginning of each record gives the reaction-time. The reaction begins with a sharp horizontal movement to the right. This is followed by the slow pursuit swing, which is more or less adequate according to the nature of the disease. The præcox pursuits, nos. 36 and 41, are typical. In mild cases the hesitation to adopt the pursuit-swing is less pronounced, but it is regularly shown by straight lines somewhere in the pursuit. The maniacal pursuit shows a tendency to get ahead of the pendulum (see upper part of lines 14, 23 and 25). This tendency sometimes appears in the first positive acceleration of the pendulum in maniacal cases. It is not entirely absent from normal pursuits or from moderate depression. In the latter cases, however, it is very rare. Other modifications of the pursuit are suggestive, but at present they permit no generalized statement.

The double breaks in each record are occasioned by the swinging of the pendulum through the recording beam of light.

that the intellectual defect is a matter of inadequate appreciation rather than a matter of attention.

The writers take the opportunity to express to Dr. H. S. Noble, superintendent of the Connecticut Hospital for the Insane, their cordial appreciation of his sympathetic interest and encouragement which made this series of experiments possible.

The appendix containing a brief account of each case by Dr. Diefendorf, together with all the experimental comparative data from the photographic records, may be consulted in original article, Brain, CXIII, 1908.

## BIBLIOGRAPHY


### Mechanical Registration of the Eye-Movements

1. Delabarre, E. B. "A Method of Recording the Eye-Movements," Amer. Journ. Psych, Vol. IX, pp. 572–574.
2. Huey, E. B. "Preliminary Experiments in the Physiology and Psychology of Reading," Amer. Journ. Psych., Vol. IX, pp. 575–586
3. Idem. "On the Physiology and Psychology of Reading," Amer. Journ. Psych., Vol. XI, pp. 283–302.

### Photographic Registration of the Eye-Movement

4. Cameron, E. H., and Steele, W. M. "The Poggendorff Illusions," Yale Studies, Vol. I, No. 1, pp. 83–111.
5. Dearborn, A. F. "Retinal Local Signs," Psych. Rev., Vol. XI, pp. 297–307.
6. Idem. "The Psychology of Reading," Arch. Philos., Psych., and Sci. Method, No. 4.
7. Dodge, R. "Five Types of Eye-Movement in the Horizontal Meridian Plane of the Field of Regard," Amer. Journ, Physiol., Vol. VIII, pp. 307–329.
8. Idem. "An Experimental Study of Visual Fixation," "Studies from the Psych. Lab. of Wesleyan Univ.," Vol. I, Psych. Rev. Monograph Supplements, Vol. VIII, No. 4.
9. Idem and Cline, T. S. "The Angle-Velocity of the Eye-Movements," Psych. Rev., Vol. VIII, pp. 145–157.
10. Holt, E. B. "Eye-Movements during Dizziness," "Harvard Psychological Studies," Vol. II, pp. 57 fol.
11. Judd, C. H. "The Müller-Lyer Illusion," Yale Studies, Vol. I, No. 1, pp. 55–82.
12. Idem. "Photographic Records of Convergence and Divergence," Yale Psychological Studies, Vol. I, No. 2, pp. 370–423, Psych. Rev. Monograph Supplement, Vol. VIII, No. 3.
13. Idem and Courten, H. C. "The Zoellner Illusion," Yale Studies, Vol. I, No. 1, pp. 112–140.

14. Idem, McAllister, C. N., and Steele, W. M. "Introduction to a Series of Studies of Eye-Movements by means of Kinetoscopic Photographs," Monograph Supplement of the Psych. Rev., Vol. VII, Yale Psychological Studies, Vol. I, pp. 1–16.
15. McAllister, C. N. "Fixation of Points in the Visual Field," Yale Studies, Vol. I, No. 1, pp. 17–54.
16. Stratton, G. M. "Eye-Movements and the Æsthetics of Visual Form," Philos. Studien, Bd. XX, S. 336.

# CYCLOTHYMIA—THE MILD FORMS OF MANIC-DEPRESSIVE PSYCHOSES AND THE MANIC-DEPRESSIVE CONSTITUTION

By Smith Ely Jelliffe, M.D., Ph.D.

ATTENDING NEUROLOGIST, CITY HOSPITAL; CLINICAL PROFESSOR OF PSYCHIATRY, FORDHAM UNIVERSITY

The history of the development of the concept of a manic-depressive psychosis is one of the most striking of comparatively modern psychiatric generalizations.

The generalization that general paresis was, in the mental realm, a disease entity, in the final fashioning of which Falret Sr.[1] played so important a part, naturally led the alienists of the times to search for other types which from the same standpoint of entity as to clinical picture might offer permanency to the then rapidly disintegrating systems of Pinel and Esquirol.

The meed of praise given to Kahlbaum for his insistence on the study of clinical pictures as a whole, and which has enriched our present psychiatric museum with such species and pseudo species as hebephrenia, catatonia, etc., has perhaps been exaggerated. Kahlbaum was far from being the first advocate of this slogan, so vigorously uttered by the present day school. Falret Sr.'s writings are many and unequivocal on this point, and antedate Kahlbaum's paper on the Grouping of Mental Diseases. In his Principles of Classification, 1860, the newer and broader standpoint is shown throughout, and in his delightful and valuable (even for the purposes of present day psychiatry) discourse on Folie Raisonnante (January, 1866) one finds him stating again and again that psychiatry has suffered too much from the following of kaleidoscopically varying symptom pictures, regardless of the clinical course.

It was this clear view of what must be borne in mind, in order to posit a psychosis of itself that led him to the amplification and

[1] Recherches sur la folie paralytiques et les diverses paralysies générales. Thèse Inaugurale. Paris, May 30, 1853.

the clearer precision of "folie circulaire" as it had been originally described by his father and Baillarger in 1854.[2]

His father and himself, writes Jules Falret,[3] have had the rare opportunity to have been able to observe in three different families the existence of this form of mental disease perpetuated in three generations; the grandmother, mother and daughter, and all with the same form.

A point of interest historically is that Falret, Jr., used the terms "mixed states" (p. 619) in his paper on folie circulaire, but gives no characterization of what he meant other than transitional periods between successive phases.

The point of present active interest concerns itself with Jules Falret's clear recognition of the attenuated types of his folie circulaire. It will prove of interest to gather here what this author says of them. Later they may be compared with contemporaneous German ideas. Falret first states that (p. 602, Études Cliniques, 1890) folie circulaire is (as defined) an hereditary affection, and generally found in a similar form in both ascendants and descendants. Speaking of attenuated forms observed in the world at large (p. 604, l. c.) he says, "In the first place there is a first category of facts which it is of importance to point out above all from the point of view of practical psychiatry and of jurisprudence. One does not often enough appreciate, and certainly one cannot too often repeat that one frequently observes, both in the family circle and in society at large individuals who are not considered as sick, even less as mentally afflicted, and whose entire lives are passed, isolated for the most part by the people who surround them in a successive round of periods of moderate excitement and of slightly pronounced melancholy, and who are in reality afflicted in an evident degree, but more attenuated, with this form of mental malady. They continue to live the life of the community, or the family life without its being necessary to treat them as sick individuals, even quite far from considering them as having a psychosis, and above all of shutting them up in asylums. So much so that, when in a period of excitement, these individuals simply appear to have changed their character, and to have

[2] See also J. P. Falret. La Manie sans delire. Thèse, 1849. J. P. Falret: Gaz. de Hop., 1851.

[3] La folie circulaire. Arch. Gén. de Med., Dec. 28, 1878, Jan., 1879.

momentarily acquired an unaccustomed activity. They occupy themselves with business; they make numerous visits, they write letters to those that they are not in the habit of visiting frequently; they have a desire to be incessantly on the move; they sleep very little, make numerous trips or projects. They take up, with feverish activity, the duties of their profession, or even take up new business schemes which they seek to advance to a state comparable with their habitual occupations. They show, on all occasions, an exaggerated gaiety. They show themselves to be intelligent, loquacious, and even spiritual, and although there may be always present great disorder in their acts, and a certain disconnectedness in their speech, those who have not known them for some time, or those who have not observed them at other times, are unable to judge of their true mental situation, although the diseased nature of this state does not escape an attentive observer, and is often appreciated with exactness by the members of their families or by those who live habitually with them.

This diseased character then manifests itself otherwise when after a more or less prolonged period of excitation, which has passed for a simple change of character, there supervenes little by little, or all at once in those individuals who, up to then, had shown gaiety and an exaggerated activity, a state precisely the reverse, to such a degree that one would believe they had to do with two different individuals. Instead of showing this exuberant activity which seems to feel neither fatigue nor the need for rest, these patients cease to go out, to make calls, to do business. They change their character completely: they become sedentary, incommunicative, almost mute; they flee from the world, seek solitude and isolation; speak little, or reply briefly to questions addressed to them, complain of general malaise, of a very sad state of suffering, of præcordial anxiety, of loss of appetite; they are sad, unhappy, anxious without reason, or for slight occasion. They are conscious of their own condition and regret it, but cannot be brought to modify them; they thus come to acquire a distaste for living, to refuse food, and in extreme cases, they shut themselves up in their rooms for several months; without attracting in a notable manner the attention of those about them, especially when one knows that they are the subjects of what one vulgarly calls "black humors," "the blues."

As to the public, they see these people only from time to time, and have no occasion to see them when they are shut up at home; they cannot doubt the diseased state in which they are found for several months, and when they see them appear later at a time when the period of excitation surges up, they then refind them such as they had known them before. They think of them as eccentric characters, gay and lively, and of feverish activity such as they have observed with certain individuals, but they do not suspect the existence of a morbid state which is appreciable only by the successive reproduction of periods of excitation and of depression which the world is not called upon to determine.

Such is the lightest and the most often overlooked phase of folie circulaire, the observation of which rarely obtains within hospitals for the insane.

Falret then discusses the severer grades of folie circulaire, such as are observed in the asylums.

That psychoses showing periodic attacks have been recognized for centuries will admit of no question, yet I cannot permit this opportunity to go by without calling in question the hasty generalization that has been widely spread, and supported by so good a student of historical problems as Farrar,[4] that Aretaeus was really the father of manic-depressive insanity. Aretaeus never dreamed of anything like the modern conception. This no one will doubt, and only by reason of a definite misinterpretation of the words "mania" and "melancholia," and a failure to realize what these words meant to writers of the early centuries, has permitted such an erroneous impression to originate.

In a short note on Hippocratic psychiatry,[5] I have again called attention to what has long been known, that the ancients did not use the words mania and melancholia in any sense as we now employ them. Melancholia was not at all synonymous with depressive conditions as a whole. It was used to designate at times the wildest excitement, and mania was used more as synonymous with insanity, craziness, etc., than it was with excitement. Thus, for instance, the word, used so frequently by Aretaeus, mania-melancholia, is interpretable only as a melancholic insanity, and might have included some excitements as well as some depres-

[4] Some Origins in Psychiatry, Am. J. of Insanity, 1909.

[5] Alienist and Neurologist, Feb., 1910.

sions. In the passages so frequently quoted as substantiating the claim that Aretaeus noticed the periodic change from mania to melancholia, it strikes me it has been entirely overlooked that he is discussing the vexed question of the relation of hypochondria to a depressed melancholia, and all that his descriptions really say is that a hypochondria passes over into a depressed melancholia. So much for the early analogies with the idea of manic-depressive insanity. I shall hope to discuss this more fully later, but any one who will read Arnold's historical notes, particularly as applied to Aretaeus, will perceive that he at least had not been misled into assuming that the ancients used the symbols mania and melancholia as we use them to-day.

But this is a pure digression into the historical realm. I wish to assume the reality of so clear a syndrome as to permit us to posit the belief in a manic-depressive psychosis. Not that everything now thought of as belonging to this psychosis will ultimately find a resting place there, but that there is a nucleus in the constellation that is a mental disorder in as definite a sense as paresis is a disorder, or that there is a dementia præcox nucleus as well.

Personally the word cyclothymia seems to be a useful one, not perhaps strictly in the sense as first proposed by Kahlbaum, but as a concept expressing one of two things, or both; namely, mild grades of the manic-depressive psychosis, and the constitutional features that underlie such personalities. In the former sense it has received much attention from the hands of modern writers, particularly Willmans, and in the latter has achieved some popularity in France at the hands of Deny, and more particularly by Kahn.[6]

The work of Falret, which can never be overlooked in the history of the development of the ideas of the manic-depressive psychosis, cannot now be reviewed, but a brief glance at Kahlbaum's original paper may be of interest. Kahlbaum, it will be recalled, had already given the concepts hebephrenia and catatonia somewhat as in their modern cast. In his paper on cyclical insanity[7] he adopts Falret's idea of a cyclical psychosis as one of the most settled features in psychiatry, comparing it in definiteness with paresis and epilepsy. He speaks of it as extremely

[6] Cyclothymia, 1909.

[7] Ueber cyklisches Irresein. Breslauer aerztliche Zeitschrift, 4, 1882.

common, and as occurring also in such mild grades as rarely to come to the attention of asylum physicians. He defines cyclical insanity in the conventional manner of periodic attacks of excitement and depression, following one another with real or apparent sound intervals, and with a certain uniformity in the symptom picture. He calls attention to the apparent anomaly of the alternating or periodic occurrence of two such strikingly different types of mental disorder as a part of what apparently is a single disease process, marked by a definite chronicity. He then speaks of these, not as two separate disease forms, but as two stages occurring in the same disease. Kahlbaum (p. 218, l. c.) is inclined to believe that these manic and melancholic stages are different from what he would term ordinary or classical manias and melancholias, and gives some interesting differentials which need not detain us at present.

The manic phase of cyclical insanity differs even more from classical mania than does the depressive phase from melancholia. The folie raissonante of the French expresses best the manic phase of the disorder.

After a somewhat lengthy disquisition on philosophical principles, Kahlbaum returns to the question of prognosis, and on the basis of a relatively good prognosis on the one hand and the development of a secondary dementia on the other, he would divide the cases of cyclical insanity into two groups, which really should be considered as things quite different one from another. On the one hand one has a partial disturbance of the mind, a primary disorder of the feelings, a true emotional disorder, the other is a total disorder, affecting all three portions of the soul life, intellect, will, and feelings, and results in degeneration. Vesania typica circularis he proposes to call the latter, and he coins the word cyclothymia (Cyclothymie, p. 221, l. c.) for the former.

A true depression remaining as such, and not a depressed phase of a circular insanity, he terms with Fleming a dysthemia, while a manic state is a hyperthemia. Thus the simple emotional psychoses are dysthemia and hyperthemia, and their combination a cyclothymia. Kahlbaum apparently never developed his ideas further, and gives no detailed description.

Hecker, Kahlbaum's follower, seemed to take up the same task as he undertook in the case of hebephrenia, namely, that of popu-

larizing his teacher's early descriptions, for some sixteen years later in a paper on Die Cyclothymie, eine circuläre Gemüthserkrankung,[8] he adopts Kahlbaum's term and discusses its symptomatology, differential diagnosis, and treatment.

Hecker first acknowledges his indebtedness to Kahlbaum and accentuates the nondementing feature that should characterize cyclothymia from others of the periodic psychoses. Cyclothymia is solely an alternation of a dysthemia and a hyperthemia indicating variations in the emotional tone of the patient with intact intellectual faculties. Here it may be noted that Hecker calls attention to Kraepelin's tentative adherence to Kahlbaum's general thesis.

Hecker makes the acute observation that whereas the depressive phases in the cycle fall under the ban of suspicion as psychotic, it is only rarely that the milder excited periods are recognized as pathological. Hence the astonishing number of periodic depressions which are noted and described in psychiatric literature, but a dearth of periodic mild excitements. He writes that in ordinary practice they are diagnosed as neurasthenics. At present we find them largely parading under the more modern symbol psychasthenia. The patients are for the most part treated for their fancied physical ailments.

The chief and fundamental symptom of the depressive stage of cyclothymia is the psychical retardation with absence of all delusions or hallucinations associated with an intense and definite, even though not always correct, insight. The patients complain, in the first place, and continuously, of their loss of ability to do mental work. They have the feeling that they will never be able to do any more work, and that they do everything upside down; of their indifference to persons and things which had been of great interest to them. They describe their condition as an inward hardening, a stoniness, as though there were a curtain, a wall, or what not between them and the world. Every resolution is difficult, all action an affliction. They must be pushed to accomplish anything; prefer to be left alone, and reject the overtures of friends because it may be necessary to talk to them. Many wish to remain in bed all day in order to be relieved of all duties. Others again, in spite of their internal conflicts, are able to so comport themselves before the public as to avoid notice. When

[8] Zeitschrift für praktische Aerzte, 7, 1898, p. 6.

such begin to express their troubles they are usually regarded by those about them as imaginary. This judgment is all the more often made by the laity, especially when some symptoms of excitement are interspersed with the depressed ones. One symptom in particular is noticeable by reason of its contrast with the patient's complaints of apathy and indifference; this is a very striking tendency to and capacity for criticism. The patients see everything and feel a thousand little things as inconvenient and disturbing quite in contrast to the true melancholic. They complain of incompleteness in the arrangement of the rooms, complain of the food, of the service, often not without foundation, but quite in contradiction to their expressed state of indifference to the world at large.

Hecker further states that it is extremely common for these patients to occpy their minds with suicidal ideas, and that frequent talking about it seems to afford some relief. The whole condition is often likened by the patient to a machine whose oil is dried up, while in the contrasting state the patients speak of the machine as too well lubricated.

This latter state, which Hecker says Kraepelin terms "hypomania," and Schüle "mania mitis," or "mania mitissima," which he observes are to be considered as more fully developed forms, shows a marked contrast with the former state. The patients feel themselves the sense of well being, they are wittier, cleverer and more capable than on healthy days. The tendency to fault finding, which, as has been seen, is present in the depressed phase, is strikingly amplified or modified. These patients often have a very scornful, mocking attitude, and the internal unrest leads to more or less exaggerated activity. Not only can these patients stand more continuous work than on their well days, but not infrequently is the character of the work a great deal better. Often those of minor musical talent rise to heights rarely reached, and in matters of artistic merit others reach a higher level than ever. Some show a skill in literary production rarely equalled in their normal phases. The biography of the "Mind that Found Itself" is an evidence of this cyclothymic activity after a recovered cycle of a marked depression and manic cycle in an acute psychotic outbreak.

Life is seen now only on its rosy side. The desire to help every-

body and interest themselves in everything is manifest throughout. Much philanthropic overactivity and many reform propaganda receive their greatest stimuli from minds in this hypomanic stage.

Hecker relates an instance of multiple marriage engagements made in the euphoric state and broken in the depressive phase in a patient with cyclothymia.

In the mild stages, patients with cyclothymia are regarded as normal. So soon, however, as the disorder develops a little more, as it is apt to do in some, if not all, of its attacks, a series of more striking symptoms arise. There is a great tendency on the part of the patients to be extravagant and careless in their purchases, a tendency to run about and to peculiar actions which the patients themselves reason about with keen relish, an abnormal sense of the ego, the wish to push oneself forward constantly, an exaggerated desire to bedeck oneself with badges or other evidences of accomplishment, medals, ribbons, buttons, etc. Hecker notes that not infrequently patients in this state have been mistaken for paretics, a mistake which the writer has had occasion to observe three times in the past six months.

In certain patients, Hecker writes, one finds the abnormal development of lying and drinking tendencies and the desire to frequent resorts of a questionable character.

## Symptoms

I do not purpose to discuss the symptoms of the more frank manic depressive psychosis, but wish to call attention to some of the signs, at times slight in themselves, at times appearing isolated —again in combination, which are indicative of the manic-depressive make up, and of the milder types of this psychosis, to which we have thought to give the name cyclothymia originally proposed by Kahlbaum.

As to the former, the signs of cyclothymic constitution, there is much to learn. Our casuistic contributions as yet are meager, and offer only tentative and suggestive material, rather than fundamentals which admit of no controversy. As I purpose to take this matter up in a separate communication I shall omit its further consideration at this time.

In the definite depressive phase of the manic-depressive psychosis, it is well known that complaints of physical ailments are extremely common and persistent, but the mental picture is so apparent that the foundation for the belief in the physical illness is recognized at its true value, and rightly dismissed from the foreground in the therapeutic attack. This, however, is not the case with the cyclothymic. Here every effort is made to conceal the mental disturbance, to minimize it, and as a corollary one finds a corresponding enhancing of the complaints of physical distress. The depressed cyclothymic hides his mental trouble because he has insight, and does not desire to be considered mentally ill, and thus pushes the physical into the foreground, perhaps himself believing it to be the source of his depression of spirits.

Such patients may be seen at any and at all times in the consultation rooms of the gynecologist, the laryngologist, the ophthalmologist, the internist, and above all the gastro-enterologist. These patients are not the false gynopaths, the false cardiopaths, the false gastropaths, and enteropaths of Dejerine—at least many of them are not—these make up still another category, but are true cyclothymic cases in the depressed phase. They are treated for weeks, or even months—get well—swell the statistics of favorable action of this or that operation, this or that remedy, not to mention the coffers of the enthusiastic, but blind specialist, and then go through another series of gynecological tinkering, of nose and throat sprays, of refitting of glasses, of stomach washing and of intestinal medication. Some of these cyclothymics never pass out of the mild class of attacks and their affection is rarely recognized. With others, however, the onset of a more severe manic or depressed attack affords the clue to the interpretation of the whole process.

In a restricted sense every mental disorder is a disorder of the entire organism since every organ of the body has a definite cortical representation. In the cyclothymic this relation of the brain to the somatic organs is strikingly illustrated and both vasomotor and secretory anomalies are almost constant accompaniments. The cessation of the menses, the diminution of the salivary and renal secretions, and disturbances of the intestinal canal are among the most striking of these abnormalities, and not enough

weight has been put especially upon the latter. I can take up only a few of these types in the restricted time at my disposal.

*Gastro-enterological Types.*—In the vast majority of cyclothymic attacks in the depressed phase, there are present very definite and tangible disturbances of the gastro-intestinal tract. The so-called nervous dyspepsias are in great part cyclothymic, and it behooves our gastro-enterologists to recognize such forms and desist from useless therapeutic attempts. Kahn has likened these attacks to a prodromal aura. There are many classical illustrations in Raymond and Janets' works, where these cases are described under the diagnosis of obsessions, neurasthenias and psychasthenias.

These cyclothymic digestive disorders are not stereotyped. Constipation is usual, and some slight dulness of mind, with persistent dull headaches and frequently restlessness and sleeplessness, although sleep disturbances seem more pronounced in those attacks which show a slightly manic tinge. In the depressive attacks which usually pass under the head of nervous dyspepsia[9] —neurasthenia and the like, the mood is usually plaintive or distinctly cast down. Effort is largely automatic, and lacks spontaneity. The sense of being driven to do one's work is marked, although a fairly high degree of efficiency may be present. Only the necessary things are attended to, and some of these neglected.

The receptivity is much diminished. Emotional indifference is frequent. These patients recognize their laziness, and often take refuge behind sententious philosophy—"that life is hardly worth the candle"—"God is unjust"—but they rarely weep, nor show any marked anxiety, although timidity may be present.

These mental symptoms they try to hide and instead speak of their continued loss of appetite, their diminishing weight, the bitter taste in the mouth, the acid eructations. They feel pressure in the body, a sense of stiffness and occasionally have profuse diarrheal discharge following marked obstipation. They have marked anxiety and believe that they are suffering from ulcer of the stomach, or that carcinoma is present. Careful and tactful suggestion often reduces the anxiety, and relieves the patient for a time, but the symptoms show a marked tendency to

[9] Dreyfus: Die nervöse Dyspepsie, 1908.

remission in spite of negative organic findings with the possible exception of a markedly reduced motility, which is a result of the mental depression.

Flemming, Schröder van der Kalk, Greisinger, Kraft-Ebing, Schüle, Alt, and most of the older psychiatrists, regarded the stomach disturbances as primary in just this sort of case, whereas it seems now with the clear definition of manic-depressive insanity before us that the very opposite is the real situation. These patients get well not because of the local applications or the local therapy, but because the cycle has run its course or that proper mental therapeutics has been applied. Some of the most striking cures—so-called—are seen in disciples of osteopathy, and kindred sects.

*Dipsomaniacal Types.*—The cyclothymic constitution and mild attacks of cyclothymia manifest themselves very frequently under the guise of periodic alcoholic debauches. In the former case the alcoholic excesses are apt to be fairly short in duration, and are often interspersed with periods of productive energy, often in the gifted of a very high order of efficiency. In the more pronounced cyclothymic attacks the debauches have a tendency to be much more prolonged, but as has already been intimated, hard and fast lines are not to be drawn between the attacks which may be regarded as purely evidences of the cyclothymic constitution or those of a more frank outbreak of a definite psychosis.

Illustrative types of this form of so-called dipsomania are not difficult to find. They are frequent in the general practitioner's work, although their relationship to a well defined psychosis in a minor type are overlooked, precisely as walking typhoids may be disregarded. Asylum studies are usually silent regarding this type of case. General literature and history abounds in references to this type of phenomenon, the best illustration of which perhaps is afforded in the life of Alfred de Musset, as told by his brother, Paul de Musset. "At times[10] at the bottom of an armoire he had an old yellow box coat, with six mufflers, and which could be wrapped about him three times. Thus muffled up, he would lie down on the floor (tapis) of his room and hum in a lamentable

[10] Paul de Musset. Bibliographie d' A. de Musset. Charpentier, 1879, p. 91.

tone some old contemporaneous air. Then in the evening he would cast aside these rags and put on his best clothes. This change of decoration was sufficient to turn the course of his ideas; he would leave to make a tour of the cafés of Paris where the pleasures of the world made him forget the reverse of fate.

Soon he would be in a fever of excitement. . . . One spring evening, says Paul de Musset, on returning from a walk, Alfred recited to me the two first couplets of a dialogue between the muse and the poet (La nuit de Mai) which he had just composed under the horse chestnuts of the Tulieries.

He worked without interruption until the morning when he appeared at breakfast. I did not notice any signs of fatigue on his face. He had as his Fantasio the month of May. The muse possessed him. During the day he would take the lead in conversation, and work like a chess player who plays two games at one time. At times he would leave us to write a dozen verses, and then would return to chat with us. But at night he returned to work as he would to a lover's rendezvous. He had late supper served him in his room. He purposely asked for two services, in order that the muse should have her place designated.

All of the lights were lit. He lit twelve candles. People of the house seeing this illumination would think he was giving a ball. On the morning of the second day, the piece having been accomplished, the muse took herself away, but she had been so well received, that she promised to return. The poet blew out his candles, went to bed and slept until evening. On awakening he reread the verses, and could find nothing to retouch.

After the inspiration—daughter it may be of excitation—here is the other stage. Then from the ideal world in which he had lived for two days, the man fell brusquely to earth, sighing as if one had awakened him from a delicious and fairy-like dream.

After the enthusiasm there followed all at once an ennui, a distaste for ordinary life, and from its petty miseries, a deep melancholy. In order to relieve himself from such a depression, it seemed that all the luxury of Sardanapolis, all that Paris could offer of distractions and of refinements could hardly suffice."

And then Paul de Musset adds—"in the eyes of most people

these alternations of over-excitement, and of depression are only weaknesses, this is an error."[11]

Georges Sand[12] has given us new testimony of these crises of moods (humeur).

"They were together in Italy, he wished to work, but suddenly he felt himself struck with a momentary loss of power, and fell into one of those cases of spleen against which he did not know how to react alone. He would be overcome by emotions coming from without; magnificant music came from the ceiling; an Arabian horse would come in through the key hole. It made no difference what delicious and terrible occurrence which would tear him from himself and under the impulsion of which he felt exalted and renewed. But here is the other period.

The days following he did not come home at all at night—he went out he said—in a boat and exercised himself rowing and taking lessons from a local fisherman. He pretended to find that the fatigue, which lessened the excitation of his nerves, was good for the work of the afternoon. But this excitation did not constrain him to spend the entire night in the boat of some fisherman. And then Georges Sand makes the sorrowful disclosure of the dipsomania of de Musset.

"Has he not said to me, and alas, almost proved it, that I smothered his genius in wishing to destroy his fever. When I believed him to have come to the limit of disgust in his excesses, have I not seen that he was anxious for more? When I have said to him return to the world, he feared my jealousy, and threw himself into gross and mysterious debaucheries. He would come home drunk, with his clothes torn and his face bleeding."

Is it not a matter for reflection to learn that the same morbid symptom may show itself under the aspect of a night of orgy, or of a night of sublime inspiration, says Kahn.

It is, I believe, recognized by many at the present time that dipsomania—or periodic drunkenness—is not by any means always an epileptic equivalent. Such is by far too narrow an interpretation, and, in fact, I am inclined to regard periodic drunkenness occurring as an epileptic equivalent to be extremely uncommon. That such cases do occur is unquestioned, and they are

[11] L. c., 145 and following.

[12] Elle et lui, p. 56, Levy.

not frequent, but on the other hand, periodic drunkenness is a common cyclothymic manifestation, as well as a frequent complication in a fully developed manic-depressive psychosis. I have observed it both in the cyclothymic depressed, and in the cyclothymic excited periods—though rarely in the same individual. Its occurrence in some cyclothymic mixed states has been commented on by Dupré,[13] and such an interpretation has seemed to me justifiable in a few cases under personal observation.

My own experience has been too limited to offer any figures as to the tendencies, but I have seemed to encounter considerable quiet drinking which has brought many a patient to the verge of an alcoholic neuritis, and even a Korsakow syndrome in the depressed cyclothymics, whereas the more boisterous and active debauches are found among the hypomanics.

*Sexual Types.*—Closely allied with the subject of periodic drinking, and often, though not always, accompanying it, the subject of periodic sexual erethism and sexual frigidity asserts itself.

The acute mental disturbances of young brides, and the periodic apparent hypomanic disorder found in fiances are not here referred to, Romfeld[14] and Dost[15] have discussed these questions fully, but I refer to shorter or longer manifestations of abnormal sexual excitement and of sexual frigidity which are the expressions in the sexual sphere of cyclothymic attacks.

It is far from uncommon to learn that the loss of chastity and conception has resulted to a young woman whose self-control was reduced by reason of her mental disorder, mild though it may have been. Such a distressing complication of a cyclothymia occurs in some of the "best families." Again hasty, and sometimes ill-considered marriages have been permitted because of excessive sexual excitement in some young woman which excess has been the expression not of a balanced ardent nature, but of a pathological state, with all the ear marks—for one who can read the little things—of a cyclothymia. Such marriages continue to supply their later stock of those not only with a cyclothymic constitution, but with children who develop a well-marked manic-

[13] Ballet, Soc. de Neur. de Paris, July 5, 1900; March 7, 1907. Soc. de Psychiatrie de Paris, March 30, 1903.

[14] Zyclothymie bei Brauten. Med.

[15] Allge. Zeits. f. Psych., 54, 1902.

depressive psychosis. Illustrative cases can be supplied by practically all the members of this association.

With this short introduction to the general topic of the mild types of manic-depressive psychosis, or cyclothymias, I leave the subject for your consideration.

## LIST OF PAPERS READ BEFORE THE NEW YORK PSYCHIATRICAL SOCIETY

April 1, 1903.
DISCUSSION: "Infantile Insanity in its Relation to Moral Perversion and Crime."
May 20, 1903.
DISCUSSION: "On the Classification of Mental Diseases."
November 4, 1903.
No paper.
January 6, 1904.
PAPER: "Plans for Psychopathic Wards and Hospitals." Dr. Clark.
March 2, 1904.
DISCUSSION: "Report of the Committee on Classification of Mental Diseases."
May 4, 1904.
PAPER: "Fright as the Cause of Mental Disturbances." Dr. Bailey.
November 2, 1904.
PAPER: "Curability of General Paresis." Dr. Dana.
January 3, 1905.
PAPER: "Habit Disorganization in Essential Deteriorations." Dr. Meyer.
March 1, 1905.
PAPER: "Mental and Nervous Diseases in Classic and Pictorial Art." Dr. Clark.
PAPER: "Art Among the Insane and Degenerate." Dr. Dana.
May 3, 1905.
PAPER: "The Mechanisms of the Phenomena of Psychopathology." Dr. Hirsch.
October 4, 1905.
PAPER: "Drug Deliria." Dr. Hoch.
December 6, 1905.
PAPER: "Insanity as a Result of Hysterectomy and Oophrorectomy." Dr. Hammond.
January 6, 1906.
PAPER: "People vs. Wood." Dr. Bailey.
March 7, 1906.
PAPER: "People vs. Young." Dr. Hirsch.
May 2, 1906.
PAPER: "Plans and Policy for the Work of the Coming Winter." Dr. Meyer.
November 7, 1906.
PAPER: "Diagnostic Criteria of General Paresis." Dr. Campbell.

January 2, 1907.

PAPER: "The Prognostic-symptomatic Complex of Manic-depressive Psychosis." Dr. Kirby.

March 4, 1907.

PAPER: "Psychogenetic Factors in some Paranoic States." Dr. Hoch.

May 1, 1907.

PAPER: "Recommendations Concerning the Improvement of Medico-legal methods." Dr. Bailey.

November 6, 1907.

PAPER: "Ocular Disc changes in Dementia Præcox." Drs. Clark and Tyson.

PAPER: "Anxiety Psychoses." Dr. Kirby.

January 8, 1908.

PAPER: "Psychogenesis and Dementia Præcox." Dr. Hoch.

March 4, 1908.

PAPER: "The Relation of Hysteria and Psychasthenia to Dementia Præcox." Dr. Meyer.

May 6, 1908.

PAPER: "On the Voluntary Admission to State and Private Institutions." Dr. Brooks.

November 4, 1908.

PAPER: "A Study of the Mental make-up in different Functional Psychoses." Dr. Hoch.

January 6, 1909.

PAPER: "Etiological Factors of the Psychoses." Dr. Mabon.

PAPER: "Racial Psychopathology." Dr. Kirby.

PAPER: "Ocular Reactions among the Insane." Dr. Diefendorf.

March 3, 1909.

PAPER: "Multiple Melancholia." Dr. Dana.

PAPER: "A Comparative Study of the Capacity for Mental work in Dementia Præcox and Alcoholic Insanity." Dr. Cotton.

May 5, 1909.

PAPER: "Clinical Forms of Periodic Drinking." Dr. Bailey.

PAPER: "Contribution to the Etiology of Manic-depressive Insanity." Dr. Hoch.

November 3, 1909.

PAPER: "Further Report on Cases of Alleged Cured Pre-paresis." Dr. Dana.

PAPER: "On some Ethical questions in Psychiatrical Expert Work." Dr. Hirsch.

January 5, 1910.

PAPER: "The Alleged Increase of Insanity." Dr. Stedman.

March 2, 1910.

PAPER: "The Content and Form of the Psychosis or Psychoanalysis in Psychiatry." Dr. Campbell.

PAPER: "The State Care of the Dangerously Insane." Dr. Hammond.

April 27, 1910.

PAPER: "A Case of Malingery." Dr. Cotton.

November 2, 1910.

PAPER: "The Insane in Japan." Dr. Peterson.

PAPER: "Cyclothymia,—the Mild Forms of Manic-depressive Psychosis." Dr. Jelliffe.

# THE EYE SYNDROME OF DEMENTIA PRÆCOX

## Ocular Signs and Symptoms of Dementia Præcox and their Significance, as Observed in 115 Consecutive Cases[1]

By H. H. Tyson, M.D.

SURGEON, N. Y. OPHTHALMIC AND AURAL INSTITUTE, NEW YORK CITY

AND

L. Pierce Clark, M.D.

SENIOR ATTENDING PHYSICIAN, HOSPITAL FOR NERVOUS DISEASES, OF NEW YORK CITY, NEW YORK

In 1899, Seglas saw a case of anxious melancholia, followed by mental confusion, evolve parallel with gastro-intestinal auto-intoxication. Meyer reported five similar cases. The idea that dementia præcox is an autotoxic disease originated with Morro, who first connected hebephrenia with this cause in 1900. The evidence for its autotoxic nature may be grouped as follows: (1) There is a coincidence of certain ocular symptoms with gastrointestinal autointoxication similar, in many aspects, to those seen in typhoid, lead colic, and simple intestinal putrefaction. (2) The urine in dementia præcox shows very defective elimination. (3) Fully one-half of the subjects of dementia præcox die of tuberculosis. (4) The co-existence of certain toxic dermatoses, such as certain types of erythema, vasomotor paresis, with chronic gastrointestinal intoxication, is noteworthy. (5) Some additional facts are at hand, as shown in the co-existence of psychic excesses, neurasthenia, etc., with states of autointoxication. (6) The study of the blood in dementia præcox shows evidence of a toxic state.

[1] This work was undertaken at the suggestion of Dr. Clark and the greater part of the work was carried out in his service at the Manhattan State Hospital and at the Vanderbilt Clinic (Dr. Starr's service). Dr. Tyson made the eye examinations and is responsible for the detailed findings of the same. Both authors are jointly responsible for the interpretative significance of the study.

More specific evidence is at hand in Kuhnt and in Blin's work, especially the latter. In 1905 Blin published a monographic consideration of the autotoxic nature of dementia præcox. He attempted to show that the retinal changes are analogous to those seen in various acute and chronic infections. It is of passing historic interest to say that Dide and Assicot (1901) had already noted the alteration of anemia and congestion in the discs of dementia præcox. None of these various observers have made any thorough or systematic attempt to analyze the significance of the eye changes in dementia præcox.

The subject of the ophthalmoscopic changes in dementia præcox receives little attention in the most recent reference works. Our bibliographic research has unearthed but two studies in which the subject is considered. These are the general papers by Kuhnt and Wochenin on alterations in the retina in psychoses, and the work by Blin on the ocular changes in dementia præcox. Kuhnt and Wochenin[2] examined 511 cases of mental diseases with the ophthalmoscope. Of these there were 5 cases of hebephrenia. The other forms of dementia præcox are not mentioned. Pathologic changes in but one case are mentioned, presumably the others were not noteworthy. This one patient had pallor or anemia of the temporal half of the papilla.

The work of Blin is much more to the point. He examined the retina in 87 cases of dementia præcox. A second examination was frequently undertaken. Despite his extensive material, Blin appears to have made no careful analysis of the significance of the findings. Of the 87 cases, some abnormality of the papillæ was found in 59. The material was divided into three groups. Nine cases (10.2 per cent.) showed constant hyperemia of the discs, and in 23 (36.8 per cent.) the congestion was transitory. Blin does not mention the condition of the blood vessels as to overfilling, tortuosity, etc., nor does he appear to have examined closely into the coincident eye symptoms necessary to establish an eye syndrome for dementia præcox. An anemia of the papillæ was constant in 7 cases (8 per cent.), and inconstant in 15 (25.3 per cent.). He uses the term intermittent in connection with the

[2] Ueber Veränderungen der Netzhaut bei Geisteskrankheiten, Ztschr. f. Augenh., 1903, xiii, 89.

latter. There were 5 cases in which congestion alternated with anemia (5.74 per cent.).

Various points often mentioned in individual cases are not summed up, such as haziness of the border, or the absence of demarcation between papillæ and retina, predominance of lesion in one eye, and the like. Doubtless all these facts seemed somewhat contradictory and did not lend themselves easily to a summary.

Three years ago we undertook an independent research on the significance of the ocular signs and symptoms in dementia præcox. We have analyzed 115 consecutive cases. The work was undertaken with the view that a careful analysis of the eye symptoms in dementia præcox might throw some definite light on the uncertain and perplexing pathogenesis of the disorder. In this respect, we believe we have not been disappointed, inasmuch as we have found definite changes and symptoms in all cases which are fully distinctive of this psychosis.

The fundus changes as seen clinically may be divided into three groups, which are usually in the order of their occurrence, as follows:

1. Congestion of discs; hyperemia and edema; dilated, dark colored veins; slightly contracted arteries and blurring of the edges of the discs, all varying in degree. These changes constitute a low grade of perineuritis of the optic nerve.

2. Congestion of the nasal side, with temporal pallor of discs, dilated veins, contracted arteries.

3. Pallor of discs, dilated veins, contracted arteries. These changes constitute anemia and partial atrophy of the optic nerve.

One hundred and nine cases were examined with the ophthalmoscope; 55 were males and 54 females. The ages of the males were from 12 to 47 years, and those of the females were from 13 to 39 years.

All the different forms of dementia præcox were under study. While the results by form types have not been fully analyzed, we are prepared to say that the more marked changes in the eye syndrome were found in the more rapidly deteriorating types of dementia præcox.

The cases embrace those who have used alcohol and tobacco moderately or to excess, as well as abstainers. It is possible that

alcohol and tobacco have contributed toward the clinical picture in some cases. In differentiating the pathologic condition of the optic nerve in tobacco and alcohol users from those in cases of dementia præcox, one observes central scotoma in the former. The one case showing a central scotoma for red in dementia præcox gave a history of alcohol and tobacco excesses. The disc changes in dementia præcox have some resemblance to those seen in the toxic amblyopia of tobacco and alcohol. But the fundus changes above detailed are seen in all cases under study. There is strong evidence that some other potent toxin is responsible for the disc changes in dementia præcox. We do not hesitate to say that we believe the toxin is primarily a vascular poison. Its most probable source is in the autointoxication (intestinal putrefaction is almost invariably evident from clinical symptoms) from the intestines or from the liver. It is possible that a faulty metabolism from a perverted action of some of the ductless glands (thyroid especially) may be the pathogenic agent. The primary departure from the normal in the disc is in the veins. They become dilated, tortuous and darker than normal. Edema of the disc appears shortly afterward. These changes are analogous to those seen in the passive congestion of the face and hands in dementia præcox cases. All these edemas produce such lasting disturbance in the nutrition of the optic nerve that slow degeneration of the nerve fibers finally results. Thus, of the 109 cases examined by the ophthalmoscope a low grade perineuritis was found 62 times in the right eye and 67 times in the left. Temporal pallor, with nasal side congested, was found in the right eye 10 times, in the left eye 11 times. Pallor of discs was found in the right eye in 37 cases and in the left eye in 31 cases.

Inasmuch as the disc changes in the first stages resemble somewhat those seen in ordinary intestinal toxemia, we have repeatedly examined the discs in a number of cases while the patients were under active hygienic treatment of free catharsis, intestinal antiseptics, baths and dietetic regulations. A marked degree of betterment was noticed of the congestive margins of the discs, but the central edema and transitional pallor have continued. Indeed, while patients were under this treatment a general physical improvement was noticed but the mental state seemed little improved.

Coincident with the study of the changes in the papillæ, the pupils were examined in 85 cases. The changes uniformly found here were not less significant. The examinations were made in moderately light rooms with the eyes fixing a distant object. The size of the pupils varied from 3½ mm. to 7 mm., with an average of $4^{77}/_{85}$ mm., while the average of the control pupils (physician and attendants) was $3^{68}/_{85}$. An average enlargement of $1^{9}/_{85}$ mm. for dementia præcox over the normal was evident.

The light reaction was normal in 71 cases and sluggish in 14. Consensual reaction was active in 68 cases and sluggish in 17 cases.

Accommodation and convergence were active in 71 cases and sluggish in 13. Hippus was present in one case.

The sensory pupillary reflex was slightly positive in 6 cases and negative in 79 instances. The psychic reflex was slightly positive in 4 cases and negative in 85 cases. Piltz-Westphal reflex was positive in 2[3] and negative in 85 cases.

Great care was exercised in measuring the pupils on account of the tendency of the eyes to change their visual lines. One is apt to complicate the accommodation and convergence reflex with the other tests. The negative reactions of the pupil appear to be due (1) to loss or partial loss of function through defective nerve innervation; (2) defect in attention; (3) diminished apperception.

Corneal sensibility was diminished in 69 cases and normal in 17.

The visual color fields were examined in 81 cases. All were found concentrically contracted. The largest field was 30 degrees, the smallest 0 degrees. The fields were practically abolished. The average of the 81 cases was 10.6 degrees, which was a marked contraction from the normal. This may be explained partially by the inattention of those patients, diminished capacity for externalization and finally, and not least, by the edema and congestion of the optic nerve (perineuritis) in the first stage of degeneration of the nerve and by the ultimate shrinkage of the new connective tissue in the partial atrophy of the nerve.

The changes in the discs, pupils, visual fields and corneal sensibility which, when taken together, constitute the new syndrome, are all in accord with each other. In our examination of all other types of insanity, imbecility or idiocy we have found no other

[3] At first positive, later examination was negative.

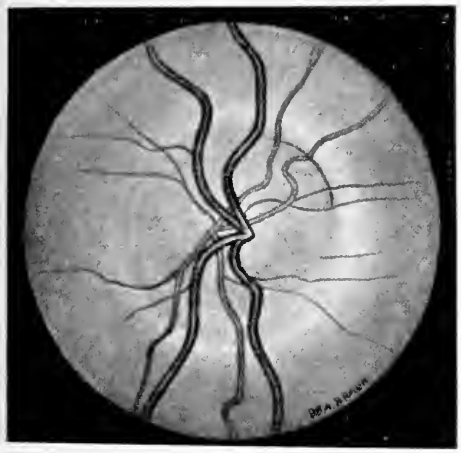

FIG. 1. A. B., "DP," 26 years old, two years in Manhattan State Hospital, Wards Island. Left eye shows congestion and edema of entire disc, edges blurred and indistinct, filling in of physiological excavation. Veins dilated, dark and tortuous; relative size compared to arteries, $2\frac{1}{2}$ to 1. (Direct method of examination.)

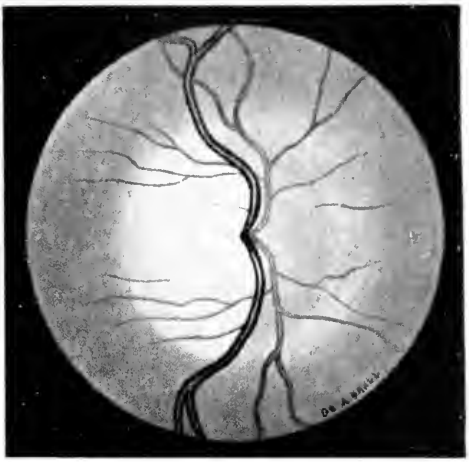

FIG. 2. K. M., "DP," in Manhattan State Hospital for 13 months. Quite deteriorated. Right eye shows temporal pallor, with nasal congestion and edema, blurring of edges on the nasal side. Veins dark and tortuous, dilated; relative size of veins to arteries, 2 to 1. (Direct method of examination.)

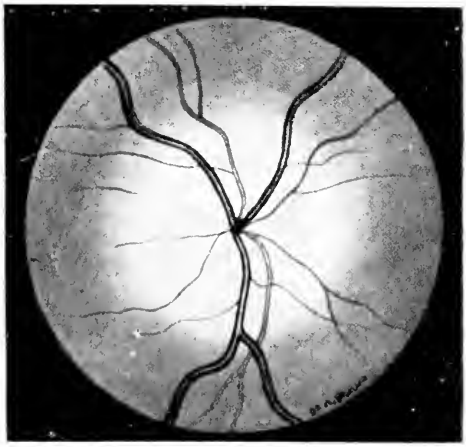

FIG. 3. M. E., "DP." Right eye. Shows pallor of entire disc. Edges blurred and indistinct. Veins dilated, dark and tortuous. Arteries slightly contracted. Relative size, compared to arteries, 2 to 1. (Direct method of examination.)

condition similar to what we have outlined here for dementia præcox.

The clinical significance of these findings is of importance:

1. They indicate that dementia præcox is attended by such an early and constant syndrome of alteration and disc, visual field, pupil and corneal sensibility as to materially aid in diagnosticating this psychosis. Consideration of the syndrome will particularly aid in the differential diagnosis of dementia præcox from the manic-depressive group, acquired neurasthenia, hysteria and the various forms of imbecility and constitutional inferiority.

2. The syndrome is a distinct contribution to the theory that dementia præcox is an autotoxic disease, and that the poison is primarily vascular, which finally induces neuronic degeneration. It points to a toxin of some sort, which is either a metabolic defect in the tissues (ductless gland defect) or, what seems more probable, that the poison is generated in the liver or in the gastro-intestinal tract itself.

3. The syndrome is of prognostic value, as the severer grades of eye changes are found in the more rapidly deteriorating cases.

4. Finally, the optic nerve lesion is quite in accord with our best knowledge of the pathologic anatomy of dementia præcox, in other tracts of the brain (than the optic nerve which itself may be counted an analogue). The early vascular changes in the brain ought to receive more serious investigation.

We desire to thank Dr. Mabon and his staff, Dr. Smith of Central Islip and his staff, and the staff of assistants in the neurologic department of the Vanderbilt Clinic (Dr. Starr's service) and Dr. C. E. Atwood in particular, for the courtesies extended to us in placing their patients at our command.

Since the publication of our original paper upon the eye syndrome of dementia præcox no similar carefully recorded observations upon the subject have been made. It is true, however, that the fundamental postulate of the cause of the syndrome, namely, the toxic character of the mental disorder, has been under continuous study. The rival camps of a psychic or somatic origin for the disease have been in a constant state of warfare. The attitude of the Kraepelinian school of somatists and the Meyer-Hoch school of psychogenists are too well known for us to detail the special viewpoints of each here. It is hoped a mutually sup-

portive middle ground may succeed the present day partisanship.

The main contention of our paper is that a fairly constant eye syndrome exists in dementia præcox, and secondly the best hypothesis for explaining the presence of the same is upon some endogenous or exogenous toxic substance. We believe the first position has been established. From the partial and incomplete nature of our study the second contention can only be urged as a contributing study to that end.

It is unnecessary to point out the remarkably low resistance of nerve-tissue to toxins in dementia præcox. As is well known in ophthalmic practice, degenerative changes in the nerve head from toxins are at first transitory, requiring time for producing distinct pathological changes as shown exquisitely in tobacco amblyopia. Even then such fundus changes are apparently slight and not recognizable microscopically but should be by more delicate methods of analysis. The extra vulnerability of nervous tissue in dementia præcox may be fairly well shown in a kindred state of inherited vice of constitution, that of imbecility. In a study of the eye in mental defectives by Clark and Cohen,[4] one of the objects of the study was to note whether a parallelism in constitutional defect, namely a strong tendency to neural degeneration, existed in both disorders. It is interesting to note in this connection that the authors found fully three fourths of all cases of idiocy of their study showed varying degrees of retrobulbar neuritis of a degenerative character. However one may search for exciting causes in the degeneration of the optic nerve in idiocy or dementia præcox, one needs to bear in mind that parallelism probably extends no further than an inherent tendency in both, for the development and character of the changes in each are totally unlike. Then, too, defectives never have the pupillary or other signs of the eye syndrome of dementia præcox.

In examining the fundus of the eye, allowance must, of course, be made for each individual variation within relatively wide physiological limits. The difference in appearance of the fundi in anemic individuals and well nourished ones is not inconsiderable. The same relative disproportion in sizes of arteries and

[4] A Study of the Eye in Mental Defectives, Journal of American Medical Association, April 16, 1910, Vol. LIV, pp. 1287, 1288.

veins exists in most all cases while the actual calibre of vessels varies greatly.

As to the different stages of the fundus changes since studying our cases over a longer period of observation, it is a question with us whether our first and second stages should not be transposed. As is well known and first pointed out by Horsley, in choked disc from brain tumor, the earliest changes are found in haziness of the superior nasal quadrant of disc. But as in the examination of the early cases examined at the clinics (prehospital or preasylum stage) we noted a majority of the congestive stage, *i. e.*, entire disc congested, etc., as originally described in stage No. 1.

Size of pupils: Those which were less than the normal average were taken in bright light and were included so as to account for and make a total average for all our cases examined, otherwise if the average size of pupils were considered only of those that had been measured in a moderately lighted room as the majority had been, then the average size would have been a trifle larger than reported.

Great care needs to be exercised in testing sensibility of cornea; one should not touch eyelashes, nor lids, nor have image of approaching objects or test fall on visual field. One should carefully approach the cornea from the periphery. Additional cases making 200 in all have been examined and about the same percentage of changes have been found as were observed in the 115 first reported.

Examination of fundi of dementia præcox cases by Dr. Holden, subsequent to our examinations, changes were noted by him in about fifty per cent. of cases; but he did not study the syndrome in its entirety. He confined himself entirely to examination of fundi alone.

The recent, careful and rather exhaustive work of Southard upon dementia præcox is worthy of more than passing notice. Southard made a careful examination of 63 brains obtained from dementia præcox patients who died at the Danvers (Mass.) State Hospital. In his investigations he laid particular stress on the "topographic idea" which had occurred to him, when, some years ago, he made an analysis of the first 1,250 necropsies of the

Danvers State Hospital and collected the lesions of different parts of the brain in card catalogue form. In a surprisingly large number of cases he found local areas of sclerosis, atrophy or aplasia. His conclusions in regard to dementia præcox are as follows:

"1. Existent evidence for the organic nature of dementia præcox is not wholly convincing, since (*a*) the cytologic changes described are found also in cases of toxic deliria and in cases complicated by severe visceral disease, and (*b*) the stratigraphic changes described are found also in senile cases without characteristic symptoms of dementia præcox.

2. Resort must, therefore, be had to the topographic idea, for the adequate exploitation of which total-brain sections, with cytologic exploration of *all* areas, are ideally necessary.

3. Random blocks of brain tissue with demonstration of satellitosis, infrastellate gliosis, or disintegration products of cell disorder will throw little light on the mechanism of dementia præcox.

4. The data of the functionalists (dissociation, sejunction, intrapsychic ataxia, and the like) are of the utmost importance as indicating the essential focality of the pathogenic process and the focal variations in its severity.

5. The curability of certain cases, the remissive character of some cases, the speedy disappearance of particular symptoms, the persistent complexity of reaction in some instances, the absence of characteristic severe projection-system symptoms, all indicate that the process is histo-pathologically mild and that the focal changes found will be found but slightly destructive or even irritative (in the sense of slight injuries readily repaired or compensated for).

6. Grossly destructive lesions of a transcortical character in Wernicke's sense might conceivably effect, *e. g.*, a permanent catatonic complex and doubtless will be found to do so occasionally; but the protean and progressive character of dementia præcox will exclude such transcortical injuries from playing a large part in the pathogenesis.

7. The focal lesions to be sought for will doubtless escape macroscopic notice in many instances, since the volume of apparatus engaged in affecting very prominent symptoms is often slight and is spread very thin in numerous areas.

8. Studies of the "soft brain" and of gliosis in epilepsy have proved, however, that even comparatively slight degrees of cortical gliosis can often be palpated at autopsy.

9. Palpable glioses of a focal or variable character combine in numerous instances with visible atrophy and microgyria, have been found in over half the series under examination, in cases regarded as clinically above reproach, and *not* subject to coarse wasting processes, focal encephalomalacia, cortical arteriosclerosis, or diffuse chronic pial changes.

10. The frequent co-existence of several foci of sclerosis or atrophy in the same brain and the microscopic observation of milder degrees of nerve-cell disorder and gliosis in regions without gross lesions tend to the conception that the agent is more general and diffuse in its action than would seem at first sight, so that future research may well demonstrate that certain instances of coarse brain wasting and even of diffuse chronic leptomeningitis belong to the group (microscopic corroboration necessary for assigning values to focal variations).

11. The microscopic examination of the residue of cases in which gross lesions or anomalies were not described, shows the same tendency to gliosis and satellitosis in numerous instances and the same tendency to focal variations from gyrus to gyrus exhibited by the gross lesion group. These findings suggest that the minor gross lesions and anomalies of several cases actually escaped notice (the protocols, though drawn up with a certain system, are by various hands) at autopsy, so that the probable actual proportion of gross lesions is 68 per cent. If microscopic evidence is resorted to, the organic proportion in our series rises to 86 per cent.

12. Several groups of cases were classified from the distribution of microscopic lesions, although the focal purity of these cases can often be brought in question from the results of microscopic examination (infrastellate gliosis and satellitosis also in macroscopically normal areas).

I. Pre-Rolandic group, including a superior frontal-prefrontal sub-group of paranoidal trend.

II. Post-Rolandic group, including (*a*) postcentral-superior parietal (sensory perceptual) sub-group in which catatonic features are the common factors; (*b*) occipital sub-group.

III. Infra-Sylvian group (too small for clinical correlations).

IV. Cerebellar group (catatonic features).

13. If these data find general confirmation, they will doubtless go far to unify discussion, since mild, variable and progressive intracortical lesions, proceeding at different rates in different parts of the apparatus, and having the peculiar distributions indicated above would explain adequately some of the contentions of the dissociationists, while remaining not wholly inconsistent with Kraepelinian ideas.

14. The frontal-paranoid correlation is in line with modern physiologic ideas, but it must be granted that the occipital and temporal regions, as elaborating important long-distance impulses, may well play a part also in paranoid states.

15. The cerebellar-catatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington).

16. The post-central-superior-parietal relations to catatonic symptoms are perhaps theoretically the most novel suggestion from the work, but here again the results are not consistent with modern physiology.

17. The topographic study of dementia præcox brains, both gross and microscopic, is commended as likely to shed new light on the pathogenesis of certain symptoms, notably paranoidal and catatonic symptoms."

These conclusions of Southard's are given at length not only as they are undoubtedly the best summary of the "topographic study" of the præcox brain at present hand, but because the lesions of atrophy and sclerosis are suprisingly analogous for that we note in the progressive degenerations in the nerve head itself. Our work might therefore be properly called a clinical contribution to Southard's "topographical histo-pathological study."[5]

[5] Our special thanks are due to Dr. Brun for his faithful drawings, in color, of the eye grounds in our cases. Drawings are a part of the original contribution.